Transactions

of the

Fortieth Annual Meeting

of the

American Academy of Ophthalmology and Otolaryngology

CINCINNATI, OHIO

September 15 to 20, 1935

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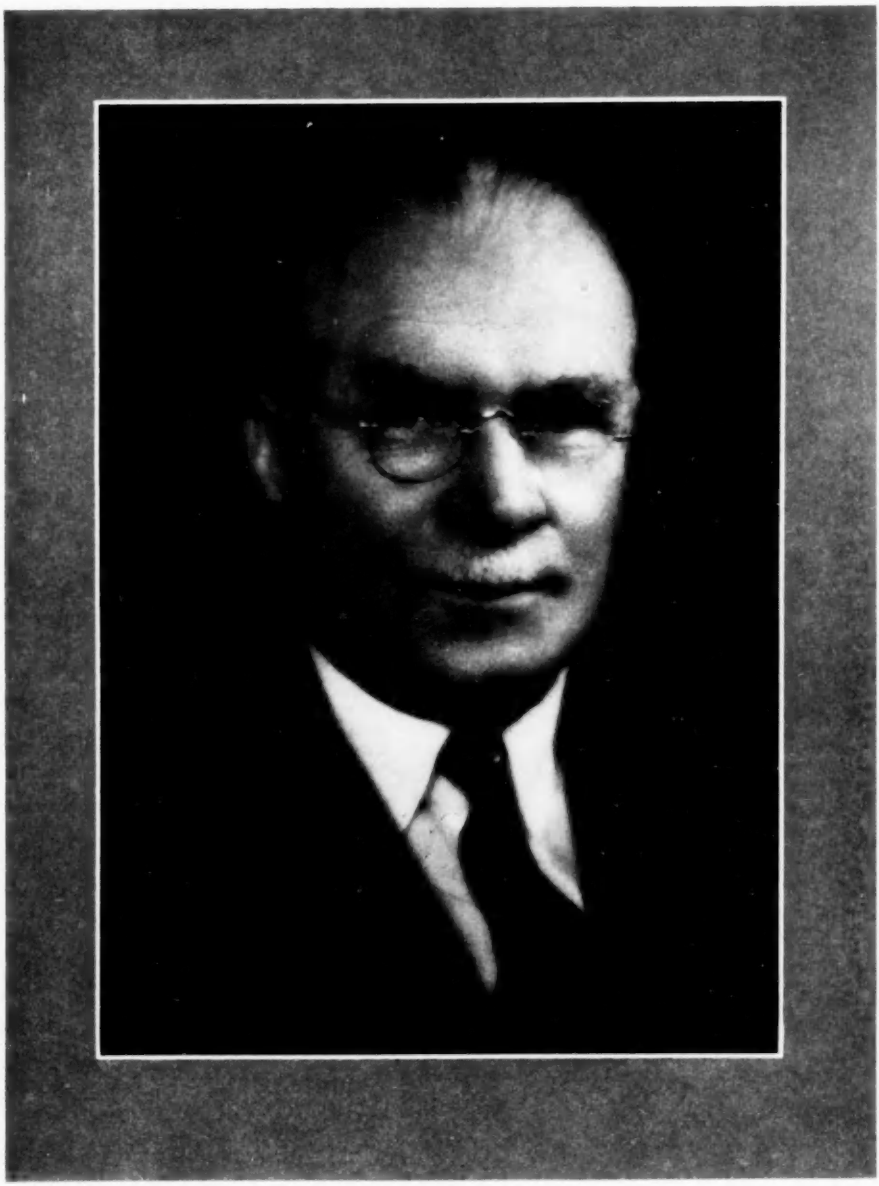

Allen Greenwood, M. D.

* * *

President of the American Academy of Ophthalmology and Otolaryngology, 1918

* * *

Dr. Allen Greenwood was born March 1, 1866, in Chelsea, Mass. He is descended from an old New England family and a direct descendant of the Rev. John Greenwood, known as the Great Separatist during the reign of Queen Elizabeth. A graduate of Harvard Medical School, 1889, he served as medical, surgical, ophthalmic and aural house officer at the Boston City Hospital for two years. He started practice in Waltham in 1890 and served several years as surgeon to the Waltham Hospital.

Early in the Spanish-American War, he went as contract surgeon to Porto Rico and became surgeon to the 19th Infantry, U. S. A. After 1898, he opened an office in Boston for the practice of ophthalmology. Soon after he was appointed assistant ophthalmic surgeon to the Boston City Hospital, later becoming surgeon and then visiting surgeon. He is still the opththalmic consultant to the Boston City Hospital. For many years he was professor of ophthalmology at Tufts Medical School.

During the World War he served with the First Harvard Unit in the British Expeditionary Forces in Flanders as medical officer, ophthalmologist, and sanitary officer. He was mentioned in dispatches by Field Marshal French for sanitary work. In the Second Harvard Unit he served as consulting ophthalmologist to the five British Expeditionary Forces hospitals located at Dannes Camiers as a lieutenant-colonel. In 1917, he served in the Surgeon General's office and organized the special hospital for head surgery, of which Colonel E. E. Ellett became the commanding officer. Dr. Greenwood wrote a book on military ophthalmic surgery based on his experiences with the British Expeditionary Forces, at the request of the Surgeon General. He organized the optical units which he took to France. In France he was commissioned as the senior consultant in ophthalmolgy by General Pershing, and organized the eye services in the hospitals of the American Expeditionary Forces in France and England. On discharge, he was commissioned in the Reserve Corps as colonel and received a citation from General Pershing.

Dr. Greenwood became consultant to the Massachusetts Eye and Ear Infirmary, the Beth Israel Hospital, Milford Hospital, Waltham Hospital, and the New England Hospital for Women and Children. For twenty-five years he has been a lecturer at the Harvard Graduate Medical School, where he started the courses in surgery of the eye. He has written several papers on ophthalmic subjects, being especially interested in glaucoma, ophthalmoscopy, and eye complications of systemic diseases.

In 1918, he was president of the American Academy of Ophthalmology and Otolaryngology. In 1920, he was chairman of the Section of Ophthalmology of the American Medical Association. He has served for more than twelve years on the American Board for Ophthalmic Examinations.

Dr. Greenwood is a member of the Massachusetts Medical Society, the American Medical Association, the New England Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, the American College of Surgeons, and the Association for Research in Ophthalmology.

* * *

The Council of this Academy has much pleasure in carrying out the wishes of the members in dedicating this number of the Transactions to Dr. Allen Greenwood of Harvard University, Boston, Mass.

Wells Hagleton

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* * *

TABLE *of* CONTENTS

* * *

Address of the President, A New Classification of Bones Forming the Skull 21
Dr. Wells P. Eagleton, Newark, N. J.

Address of the Guest of Honor, Brain Tumors and the Ear 59
Dr. Hans Brunner, Vienna, Austria

Address delivered at the Banquet, History of the American Academy of Ophthalmology and Otolaryngology 79
Dr. Max A. Goldstein, St. Louis, Mo.

* * *

SYMPOSIUM

Headache

A. Headache, From the Standpoint of the Otolaryngologist 85
Dr. Harris H. Vail, Cincinnati, Ohio

B. Headache, From the Standpoint of the Ophthalmologist 97
Dr. William H. Crisp, Denver, Colo.

C. Headache, From the Standpoint of the Neurologist 109
Dr. A. R. Vonderahe, Cincinnati, Ohio
BY INVITATION

Ophthalmologic Sessions

* * *

TABLE *of* CONTENTS

* * *

THE HYPERTENSIVE FUNDUS OCULI AFTER RESECTION OF THE SPLANCHNIC SYMPATHETICS 119
DR. F. BRUCE FRALICK, and
DR. MAX M. PEET, Ann Arbor, Mich.
BY INVITATION

ROENTGENOGRAPHIC DIAGNOSIS OF RETINOBLASTOMA 131
DR. RAYMOND L. PFEIFFER, New York, N. Y.
BY INVITATION

MASSIVE RETINAL FIBROSIS IN CHILDREN 145
DR. ALGERNON B. REESE, New York, N. Y.

OPHTHALMIC ERRORS 156
DR. HANS BARKAN, San Francisco, Calif.

MANAGEMENT OF COMPLICATIONS IN THE OPERATION FOR SENILE CATARACT 167
DR. HARRY W. WOODRUFF, Joliet, Ill.

ANISEIKONIA 185
DR. WENDELL L. HUGHES, Hempstead, N. Y.

CONGENITAL PARALYSIS OF THE SIXTH NERVE 193
DR. JAMES WATSON WHITE, New York, N. Y.

CHANGES IN REFRACTION FOLLOWING OPERATION FOR STRABISMUS 205
DR. DON MARSHALL, Ann Arbor, Mich.

STUDIES ON THE INFECTIVITY OF TRACHOMA 221
DR. L. A. JULIANELLE and
DR. R. W. HARRISON, St. Louis, Mo.
BY INVITATION

THE TRACHOMA SITUATION IN SOUTHERN ILLINOIS 229
DR. H. S. GRADLE,
DR. A. F. LENZEN and
A. M. HAYDEN, Chicago, Ill.

Otolaryngologic Sessions

TABLE *of* CONTENTS

EXPERIMENTAL AND CLINICAL SINUSITIS, RESEARCH REPORT. 241
DR. RALPH A. FENTON and
DR. OLOF LARSELL, Portland, Ore.

ALLERGY IN THE RHINOLOGIST'S WAITING ROOM 255
DR. WARREN T. VAUGHAN, Richmond, Va.
BY INVITATION

THE EFFECT OF RADIATION UPON CILIATED EPITHELIUM 269
DR. LYMAN H. HEINE, Boston, Mass.
BY INVITATION

MALIGNANT TUMORS OF THE EPIPHARYNX 281
DR. SAMUEL SALINGER and
DR. SAMUEL J. PEARLMAN, Chicago, Ill.

USE OF GELATIN PROSTHESES IN FACIAL RESTORATION 317
DR. OSCAR V. BATSON, Philadelphia, Pa.

AN EXPERIMENTAL INVESTIGATION OF TINNITUS,
RESEARCH REPORT 327
DR. W. J. MCNALLY,
DR. E. A. STUART,
DR. T. F. REID and
DR. L. H. MCCONNELL, Montreal, Canada

FOCAL INFECTION IN ARTHRITIS 351
DR. D. M. LIERLE, Iowa City, Ia.

THE RELATION OF DIET TO CERTAIN OTOLARYNGOLOGIC
PROBLEMS 358
DR. CECIL STRIKER, Cincinnati, Ohio
BY INVITATION

ANIMAL EXPERIMENTATION ON THE HEARING, ITS CLUES
TO THE PREVENTION OF DEAFNESS 375
DR. M. H. LURIE, Boston, Mass.

OBSERVATIONS ON CHRONIC INFLAMMATORY LESIONS OF
THE TRUE VOCAL CORDS 390
DR. GABRIEL TUCKER, Philadelphia, Pa.

TABLE *of* CONTENTS

Instructional Subjects 405

Presentation of Instruments and Appliances 413

Minutes of the Fortieth Annual Meeting 455

Business Meeting 461

Necrology 467

Reports 488

New Members 499

Election of Officers 504

Selection of 1936 Convention City 506

Alphabetical List of Members 509

Geographical List of Members 565

Index to Authors and Subjects 637

Joint Session

The American Academy
of Ophthalmology and Otolaryngology

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Fortieth Annual Meeting
Cincinnati • 1935

ADDRESS OF THE PRESIDENT

* * *

A NEW CLASSIFICATION OF THE BONES FORMING THE SKULL

(1) BASED ON THEIR FUNCTION AND EMBRYOLOGICAL ORIGIN; ESPECIALLY AS INFLUENCING THE KIND, COURSE AND FREQUENCY OF INFECTIONS OF THE INDIVIDUAL BONES; WITH (2) SURGICAL APPLICATIONS, ESPECIALLY AS TO THE RELATION OF OSSEOUS INFECTIONS TO MENINGITIS

WELLS P. EAGLETON, M.D.

NEWARK, N. J.

PART I

HISTORICAL BACKGROUND OF SURGICAL TREATMENT OF PETROUS APEX INFECTIONS IN RELATION TO MENINGITIS

On December 8, 1926, there entered the Newark Eye and Ear Infirmary a young girl[1] who required a single mastoid operation. Following the operation, she had pain (a) in her head and (b) behind her eye. Later she returned and another operation on her mastoid was performed, during which the surgeon noticed tissue that "looked like cheese." Forty-three days subsequent to the first operation, she developed meningitis. At autopsy, the petrous apex was eroded and filled with granulations and with caseous material without pus. I concluded that the granulation mass must be tubercular.

Two months later, on February 18, 1927, another patient had a similar history of pain behind the eye, with a running ear, but he had, in addition, a facial paralysis. At the autopsy there

1. Eagleton, W. P.: *Sixth Nerve Involvement in the Diagnosis and Surgical Treatment of Meningitis of Otitic and Nasal Origin*, I. Congress Internat. d'Oto.-Rhino.-Laryngologie, Copenhagen, 1928.

was found a similar granulomatous caries — with perforation of both the superior and posterior surface of the apex.[2]

The summer of 1927 was devoted to anatomical dissections in an effort to establish an anatomical route into the petrous apex which would allow of adequate inspection of the diseased area. The operation selected I termed "unlocking the petrous pyramid."[3] The first operation in the history of medicine of deliberately entering the petrous apex of a patient whose disease had previously been diagnosed as "suppuration within the petrous apex," was performed on June 30, 1929, after a preliminary ligation of the carotid. The patient recovered promptly, although the operation so aggravated the localized meningitis that microörganisms were temporarily free in the cerebrospinal fluid.[4]

It was an attempt to understand why certain cases of petrous apex infection cause a recrudescence of meningitis,* that led to a study of (a) bone embryology, which includes (b) *bone growth*, as well as an investigation of the (c) *relative degrees of biocellular and humoral immunity that are inherent in different tissues*. This study forced me to view the bones of the skull from a functional as well as a morphological standpoint.

And it is the factors of bone embryology, of bone growth and the reaction of different bones to infection, a localized immunity — many of these factors being well known to scientists but neglected by the clinician — that I wish to present. For a consideration of embryological and growth factors will help to explain many of the problems presented by cases of meningitis, the meningitis being secondary to the osseous infection, as well as to furnish new viewpoints for future investigations.

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2. Eagleton, W. P.: *Localized Bulbar Cisterna (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex*, case 2, Arch. Surg., 20:400 (Mar.) 1930.

3. Eagleton, W. P.: *The Cerebrospinal Fluid as an Aid to Diagnosis and Treatment of Inflammatory Diseases*, J. Laryngol. & Otol., 44:657-673 (Oct.); 44:721-738 (Nov.) 1929.

4. Eagleton, W. P.: *Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex*, Arch. Otolaryng., 13:407 (Mar.) 1931, case 2.

* V. *Acute Septicemia Cases of Apex Suppuration Associated with a Continually Positive Blood Culture and Meningitis.*
Eagleton, W. P.: *Meningitis — Result of Disease of the Petrous Apex and the Sphenoidal Basis*, Surg. Gynec. & Obst., 60:586 (Feb.) 1935.

EVOLUTIONARY, EMBRYOLOGICAL AND FUNCTIONAL BASES OF DISEASE

Life is the Great Experimenter. Invertebrates have eyes; also each invertebrate has an apparatus for equilibration, and many forms of invertebrates have mechanisms for hearing, but these are located in different parts of the body. (Figs. 1 and 2).

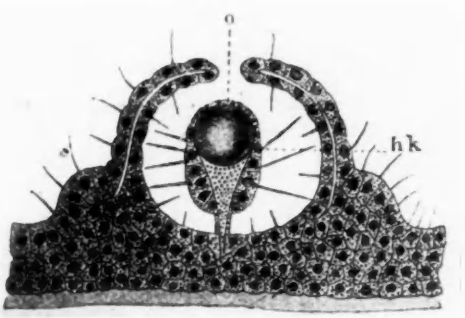

FIG. 1 — (Eagleton) — Vestibular apparatus of rhopalonema. Note delicate structure of exposed position. *(Hertwig).*

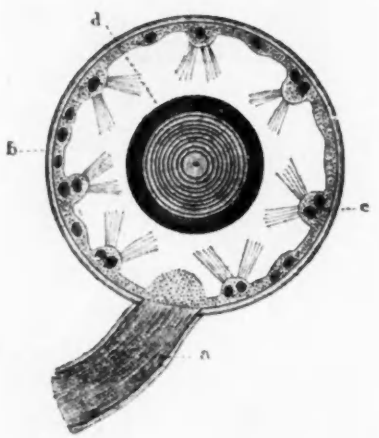

FIG. 2 — (Eagleton) — Vestibular apparatus of mollusk — much coarser and more protected because exposed to waves. *(Morat).*

However, as life evolved in the vertebrate, first in the sea, then upon the land, into the trees, and up into the air, with the localization of ganglionic masses and then a brain in the organism's forward end, it evolved with its growth special-sense mechanisms — which are, in reality, additions to its brain.

The lower vertebrates have end-organs for (1) *equilibrium*, and for (2) *touch*, and later types, for (3) *imperfect sight* (Fishes do not see well). And then as life crawled out upon the land, (4) *smell* became the dominant sense. For all running things — the rabbits, the deer — recognize their food, their mates and their enemies by *smell* and (5) *hearing*.

FIG. 3 — (Eagleton) — Brain of primitive mammal. Note large size of olfactory area — gradual growth of visual area in higher forms. *(Elliott-Smith).*

At last, when life took to the trees, (6) *vision* dominated life's acts. And with each addition to its special-sense mechanism, a provision for protection was evolved (Fig. 3). So when we reach that strange paradox, whose acts often cause us to wonder why he is called Homosapiens — the wise one, the thinking man — we find these special-senses and his brain are adequately protected against injury from forces without and from invading microörganisms within. Consequently, the bones of the skull, made up as they are for the protection of special-sense mechanisms and of neural tissue, should not be considered solely from the standpoint of their regional position, as a parietal or temporal bone, but also from that of their use as protectors and retainers of the brain, or of the special-sense organs, through which man's brain receives the external impulses that make up his consciousness.

For the bones of the skull are different; almost each fibre is different. In medicolegal trials, a small piece of cortex from a bone of a body is recognized by its shape. But a bone of the skull can be recognized by its morphology; and not only can its position in the skull be determined, but if the section be deep enough, the approximate age of the subject may be surmised with some degree of accuracy.

Functional Specificity of Tissue Depends upon Its Position as well as Its Embryonic Layer — The theory that the functional specificity of a tissue depends upon the (a) embryonic layer from which it evolves, in (b) relation to its exact position in the animal frame, has not been fully appreciated, although both are of practical as well as scientific importance.

Embryogenesis of a Bone of the Neurocranium. The Ectodermal Rudimental Radicle in the Growth of the Neurocranium — The bones of the trunk and extremities spring entirely from (a) mesoderm; that is, cartilage that forms bone in osteogenetic areas in embryonal life is composed of mesenchymal tissue, for mesoderm is the sole germinal layer in the formation of the cartilage anlage from which the bones of the trunk and extremities are formed.

However, in the skull certain cartilages and soft tissues are modified by the addition of an ectodermal quotient (Stone[5]), as during the earliest stage of embryonal growth ectoderm crawls over the head's mesodermal bed. And it is the addition of this vitalizing growth moiety that gives these bones their distinctive neurocranial character; for without this ectodermal moiety, neurocranial cartilage and some of the cranial soft-tissue do not develop (See page 41).

Thus, an ectodermal quotient — ectoderm, from which all nervous tissue is formed — becomes the vitalizing moiety of a neurocranial bone in its growth from a somatic mesodermal stratum.

Neurocranial Bones Develop by Modification of Established Stages of Somatic Bone Growth — Further, life has adapted each cranial bone to its own functional position by (a) modifying morphological patterns that had become established in the growth and development of the somatic bones[6] of the trunk and extremities. This modification life accomplished by adding to, omitting from, or combining the usual stages of bone growth differently, so that new forms of bone — a neurocranial bone — emerged from (b) alterations in usual growth processes (See page 39 and

5. Stone, L. S.: *Problems Concerning the Origin and Development of the Neural Crest and Cranial Ganglia in the Vertebrates,* Yale J. Biol. & Med., 1:7-13 (Oct.) 1928.

6. Fell, H. B., and Robinson, R.: *The Growth, Development and Phosphatase Activity of Embryonic Avian Femora and Limb-buds,* Biochem. Jour. 23:767, 1929.

40). And the specific type of growth is (c) inherent in the particular group of cells of each functionally different domain (Coghill[7]).

Thus, to grasp the significance of a bone of the skull requires a consideration of four factors:

(a) its embryological origin,
(b) its functional use,
(c) its stage of growth, and
(d) its position in relation to contained organs and surrounding structures.

While the differences in the morphology of the cranial bones are explained by variations in (e) the manner of the bone's growth in the embryo and during later life.

Periosteal and Cartilage Bone — In a very general way, there are two kinds of somatic bone: (1) *compact tissue bone, which is* (a) *formed at the periphery* by the (b) periosteum, viz., *periosteal bone;* and (2) *cartilage bone,* (a) *formed centrally* from what is called (b) a *cartilage "anlage"* — a cartilage nest.

Osseous Formation and Endothelial Protoplasmic Penetration in Bone Growth — And there are in the growth of bone two distinct growth processes: (1) *osseoid formation,* characterized by the deposit of calcium salts; and (2) *protoplasmic vascular penetration* — a reticulo-endothelial infiltration of red bone marrow into the osseous stroma; for bone marrow should be regarded as primordial vascular endothelial tissue (Lorrain Smith).

Growth in Somatic Bones — A bone's growth is accomplished by (1) deposition of, and later by (2) absorption, first of calcified cartilage and then of osseous tissue. For all bone growth is a process of (1) depositing and (2) absorption, which latter is accompanied by (3) differentiated division for function; all being perfectly synchronized, while the growth processes act both (1) in the center and (2) at the periphery.

Growth Processes in Somatic Bones — The bones of the trunk and extremities form from a combination of three processes: (1) endochondral growth, which results in both (a) cartilagin-

7. Coghill, S. E.: *Anatomy and the Problems of Behavior,* Cambridge University Press, 1929.

ous ossification and (b) endothelial protoplasmic permeation. In a long bone, central endochondral growth is accompanied by (2) peripheral periosteal bone formation; for it is periosteal bone formation that furnishes the growth increments which form the cortex encapsulating the bone. And in a manner analogous to that in which (a) an advancing proliferating new connective tissue absorbs the senescent calcified cartilage cells[8] within the central stroma, while it (b) deposits mineral salts and thus forms osseous tissue, so periosteal bone growth is accompanied (c) by an absorption of the centrally-formed endochondral osseoid elements in the interior of the bone.[9] As a result, the final structure of bone comes from bone cells furnished by the periosteum, with little of the original cartilaginous framework remaining.[10] At the same time, this vascular fibrous absorption from the periphery creates additional space in the body of the calcified cartilage for the deposition of protoplasmic (3) endothelial bone marrow, which later is evolved in the new connective tissue that has formed in the central calcified cartilage; for every Haversian canal is a prolongation of the central marrow system.

The Three Life Stages of Bone Growth — And each bone during its growth *passes through three stages* — (a) youth, (b) maturity, and (c) old age; like the rest of us, it becomes senescent — senile.

Consequently, a study of the embryological development of each bone in (a) *relation to its function, and its* (b) *method of growth, as it passes to and through* (c) *maturity and becomes senescent,* will help to explain the (1) different reactions of different forms of infection by different bones; as well as the (2) course that each type of infection will take in the particular bone involved. For one bone of the skull, the *labyrinth of the new-born babe — the neonatal labyrinth* (Fig. 4) — has passed maturity and become senescent at birth. And because it is senile, it has lost most of its vessels while the fœtus was still in the mother's womb (See page 35). And another bone, the sphenoidal basis, never grows senile. For the sphenoidal basis should be considered not simply as a supporting and mechanically protecting bone, but as an *actively functioning* organ, an organ that operates throughout the entire life of the individual.

8. Harris, H. A.: *Bone Growth in Health and Disease,* Oxford University Press, London, Humphrey Milford, 1933.
9. Leriche, R., and Policard, A.: *The Normal and Pathological Physiology of Bone.* St. Louis, C. V. Mosby Company, 1928.
10. Smith, J. Lorrain: *Growth,* London, Oliver & Boyd, 1932, p. 84.

FIG. 4 — (Eagleton) — Infant skull. Mastoid grows outward by pneumatization to protect labyrinth during first two years.

Neurocranial (Fawcett) and Facial Bones — From a functional standpoint, the skull is made up of (1) face bones and (2) neurocranial bones. The latter are bones whose position and function are dictated by their relation to neural tissue. All *facial bones* that are below the eyes are for the protection and the carrying on of the organism's respiratory and digestive activities. Facial bones have but slight relationship with the brain.

For a better understanding of their function, I suggest that *neurocranial bones* be divided into (1) *cranial vault bones*, which evolve from membrane; (2) *special-sense* (compact tissue) *bones*, which spring from cartilage; (3) *passively protective* (pneumatic) *special-sense bones*, such as the mastoid, which develop from a combination of cartilage and membrane growth; and the (4) *biocellular actively protective bones* of the base of the skull, which contain red bone marrow and continue the morphological pattern seen in the somatic bones; that is, in the ring-shaped vertebræ, the encircling ribs, the pelvis, and the long and short bones of the extremities.

(1) Neurocranial bones of the vault of the calvarium grow from membrane; they do not spring from cartilage. They are appositional or membrane bones. The internal plate of the vault bone grows from the dura outward, while the external plate forms from the covering soft parts growing inward.

The membrane bones of the vault begin their growth during fœtal life, and at birth are separated and incompletely formed. But the two large soft spots in the infant's skull gradually close during infancy. Further, throughout maturity, that is, from puberty on, the bones of the cranial vault are red marrow filled; only to lose this protoplasmic endothelial marrow during the general senescence of old age.

Surgical Anatomy of Osteomyelitis of the Cranial Vault—During growth and throughout maturity vessels run into the vault bones from the dura within and the soft part externally (Furstenberg).[11] Thus, an invasive type of osteomyelitis of the vault of the skull is apt to be accompanied by (a) a subperiosteal abscess, because of the bone's vascular supply. And furthermore, if the vitreous plate is involved, there is always (b) an extradural abscess, and frequently (c) a subdural-space abscess. Consequently, it should be a dictum that if an osteomyelitis of the skull is associated with headache, a lumbar puncture should be performed, and be the cell count high, the dura should be opened.

(2) *A special-sense bone is composed of* compact bone laminæ, which grow with the end-organ that it harbors. Special-sense bones spring from cartilage and are largely *without vessels* and for this reason they cannot be the primary seat of an infection.

Otosclerosis—Because the labyrinth is a unique special-sense capsular bone, it is the seat of a specific disease, otosclerosis, and all attempts to consider otosclerosis as a form of disease found in any other bone are destined to fail. The changes in otosclerosis are allied to osteofibrosis, but otosclerosis is not osteofibrosis, for the former is a change which occurs only in a specific bone—the osseous labyrinth—that has a specific function, the protection of the highly specialized membranous labyrinth.

In my opinion, otosclerosis is a dystrophy of that unique type of bone growth which produces a man's idiocratic special-sense labyrinth, as cancer probably is a dystrophy of all forms of tissue growth;[12] but cancer may occur in any species and in any tissue, while otosclerosis is confined to the bony labyrinth capsule of man. The loss of hearing is a secondary and a terminal manifestation of the dystrophy in the neonatal capsule's growth.

11. Furstenberg, A. C.: *Bone Regeneration in Osteomyelitic Defects of the Cranium*, Trans. 36th Annual Meet. Am. Laryng., Rhinol. & Otol. Soc., 1930, pp. 434-439.
12. Lockhart-Mummery, J. P.: *The Origin of Cancer*, London, Churchill, 1934.

Furthermore, there is no evidence that either trauma or venous stasis causes otosclerosis. And to do so, the trauma or venous stasis must occur either during fœtal life or in infancy; that is, while the labyrinth capsule is growing.

(3) *The passively protective special-sense bones are the pneumatic bones.* They grow from cartilage but have a membrane bone covering. Lateral expansion of bone by pneumatic growth in the mammal is a form of cranial bone growth. The function of a pneumatic bone is to protect its special-sense organ against external violence and infection.

A baby needs no protecting mastoid for its labyrinth, because it is not subject to external violence; thus expansion of bone by pneumatic growth only begins at birth.

On the other hand, the function of "overflow pneumatization" is for rectifying inequalities in bone surfaces that may have appeared during the development of adjacent bones. Overflow pneumatization occurs around the labyrinth and into the petrous apex during the second growth cycle — at the age of five to ten year (See page 36).

Clinical Application

(a) *Mastoiditis in Infants Is an Osteomyelitis* — During infancy the spaces between the cartilage anlage which forms the bone of the labyrinth and the surface membrane that covers it are filled with infantile fat, with the result that mastoiditis in an infant is a true osteomyelitis accompanied by severe constitutional disturbances, especially diarrhea, as first brought out by Dean,[13] because the gastro-intestinal tract, not having acquired its full functional growth, is in an unstable condition.

(b) *Mastoiditis in Children Is Usually a Local Osteitis* — After a passively protective pneumatic bone is fully developed it contains few vessels; consequently, infection is apt to be limited within the bone itself. The ordinary so-called "confluent type of mastoiditis" that occurs from childhood on can be defined as a "localized osteitis of a pneumatic bone."

(4) *The actively protective bones of the cranial base grow in utero* by an embryonic hemato-endothelial infiltration within the cellular structure of cartilage bone. Thus, the bones of the cranial base after birth contain a protoplasmic mechanism. The function of these dynamically operating organs is to protect, throughout extra-uterine life, the meninges covering the base of the brain against infection from without.

13. Dean, L. W., and Armstrong, M.: *Nasal Sinus Disease in Young Children,* Ann. Otol., Rhinol. & Laryng., 28:445, 1919.

A perfect example of *actively protecting biocellular neurocranial bone* is the *sphenoidal bases* — for, although the sphenoidal air sinus frequently becomes infected from a "cold," the red bone marrow within its spongiosa operates to overcome the microörganisms.

CLINICAL APPLICATION

(c) *Sphenoidal-Sinus Pneumococcal Meningitis of Small Vein Origin and Streptococcal Meningitis from Sinusoidal Osteomyelitis* — Thus, the number of cases of brain abscess from an infected sphenoidal sinus totals but few — about a half dozen in the whole literature, and only in two forms — (a) pneumococcal meningitis following an infection confined to the blood vessels of the submucosa; and (b) streptococcal meningitis from sinusoidal osteomyelitis. Meningitis from the sphenoidal sinus is almost always pneumococcic in origin, because the organism has a capsule and grows in the blood vessels of the bone.[14]

PROGRESS IN UNDERSTANDING OF MENINGITIS IN RELATION TO INFECTIVE LESIONS OF AN ADJACENT BONE

It has long been known that suppuration within a bone of the skull is the cause of meningitis in about 65 per cent of all cases. Fifteen years ago I demonstrated that during the initial stage meningitis is apt to be confined to a localized area,[15] and that if the fluid within the infected arachnoid cistern be early evacuated, the process is generally controlled. Some years later, I found that disease of the vessels from the bone into the meninges[16] was the cause of meningitis in at least 20 per cent of all cases. The remaining 15 per cent of cases of meningitis have been considered of unknown origin. But today I know that many of this 15 per cent are infections that have reached the meninges from the petrous apex or from the sphenoidal bases;

14. Eagleton, W. P.: *Meningitis from the Sphenoid*, Trans. 38th Annual Meet., Am. Laryng., Rhinol. & Otol. Soc., 1932, pp. 51-61.

15. Eagleton, W. P.: *The Operative Treatment of Suppurative Meningitis with Especial Reference to Irrigation of the Cranial and Spinal Subarachnoid Spaces; and Importance of Protective Meningitis from a Progressive and Therapeutic Standpoint. With an Analysis of the Cases of Recovery — Exclusive of Meningococcic — Reported in the Literature*, Trans. Am. Otol. Soc., 1921.
The Operative Treatment of Suppurative Meningitis, Atlantic Med. J., 26:575 (June) 1923.
The Surgical Treatment of Meningitis, J.A.M.A., 83:1900-1906 (Dec. 13) 1924.

16. Eagleton, W. P.: *Suppurative Meningitis of Otitic and Nasal Origin; Its Relation to Blood Stream Infection of the Pial Vessels*, Arch. Otolaryng., 15:885-905 (June) 1932.

although the infection, which is the cause of the meningitis, being within the red bone marrow, is frequently overlooked even at postmortem. This is because the red marrow within the bones of the skull's base does not react to infection by the production of visible bone necrosis and pus until days and even weeks have passed. The microscopical wont of marrow infection is illustrated in osteomyelitis occurring in a long bone of a child in whom early cutting through the cortex reveals what is apparently normal marrow (Bancroft); although the disease is accompanied by pain, high temperature and local tenderness. However, the incision usually stops the infective process.

Infection by Vascular Pathways—In involvement of the marrow-containing sphenoidal basis or petrous apex, microscopic examination reveals that infected blood vessels often enter directly into the meninges; in which case the patient is apt to succumb from meningo-encephalitis long before there is any change of bone marrow infection which is discernible to the naked eye (Fig. 5).

Thus, the whole problem of meningitis from a marrow-containing bone of the skull is largely a question of how to control an infection which passes through or from a bone by way of blood vessels.

Relationship of Vascularity of Bone to Growth and the Marrow's Dynamic Function in the Control of Infection—The somatic bones of a new-born mammal before it walks are red and soft because of (a) a free vascular supply, and because of the red bone-marrow content. But in the full-grown steer, the bone is ebony-like and composed of a firm white cortex enclosing yellow fat marrow; because during the process of growth the vessels have disappeared, the trabeculæ of the medulla have been absorbed, and the invading red bone marrow has become fat; while its periosteal cortical bone has been converted into senescent bone. A similar process occurs in a petrous apex; it grows, and when it reaches maturity, its cellular marrow turns to acellular fat.

Metaplastic Differentiation from Yellow Fat to Cellular Red Marrow—But Custer[17] has shown that in the long bones of a

17. Custer, R. P.: *Studies on the Structure and Function of Bone Marrow. I. Variability of the Hematopoietic Pattern and Consideration of Method of Examination*, J. Lab. & Clin. Med., 17:951 (July) 1932.

FIG. 5 — (Eagleton) — Hemorrhagic infiltration in basal ganglia in meningo-encephalitis.

chicken the acellular fat marrow can become cellular red bone marrow in two days, in response to the stimulus of an infection or of cancer.

This also occurs in those types of tumor of the brain that cause bone marrow reactions.

I recently examined the apices from a patient who had died of an infiltrating tumor of the temporal lobe of the brain. The neoplasm had been slowly increasing in size, with but slight vascularity, for about two years. During life he had the typical symptoms of involvement of the uncinate gyrus; he would smell a peculiar odor and then have a convulsion. At autopsy, both petrous apices were filled with mahogany-colored bone marrow, whereas normally the apices are filled with yellow fat or with air cells after about the eighth year.

Why this mahogany-colored, actively functioning bone marrow? It was the conversion of the fat within the apex into cellular marrow in response to the hemogenetic irritation of the growth.

The Apex, a Reticulo-endothelial Unit of the Base — For the petrous apex should be regarded as a local depot or unit of the reticulo-endothelial system of the skull's base; and when it is filled with red bone marrow, it reacts by hemogenetic activity, this being its response to an abnormal stimulus which has entered the circulation from adjacent tissues.

PART II

SURGICAL APPLICATIONS OF THE INFLUENCE OF THE EMBRYOLOGICAL ORIGIN AND THE STAGE OF BONE GROWTH ON INFECTION IN THE TEMPORAL BONE

Three Genetically Different Forms of Osseous Tissue Compose the Temporal Bone — The mammalian temporal bone contains two examples of the highest forms of specialized osseous architecture that have been evolved by life. It consists of three distinct parts, placed side by side, each having sprung, during intrauterine life, from a separate cartilage mass; which together make up the primordial cranial basis of the fœtus.

For the designations of the functional differences that exist between the genetically distinct forms of bone composing the temporal area of the neurocranium, I suggest the terms (see page 28):

(1) Passively protective special-sense (pneumatic) bone (of the mastoid).

(2) Special-sense (compact) bone (of the labyrinth).

(3) Actively protective somatic (spongiose red marrow-filled) bone (of the petrous apex portion of the cranial basis).

The Three Stages of Bone Growth that Are Present in the Temporal Bone of the New-born Child — Not only is the os temporale of the new-born child[18] made up of (1) three varieties of neurocranial bone; but each is in a (2) different stage of bone growth.

18. Cheatle, A. H.: *Surgical Anatomy of the Temporal Bone*, London, J. & A. Churchill, 1907, pp. 16-17.

(1) *The fat-filled, passively protective special-sense rudimentary mastoid* domain is placed superficially; its lateral extension outward by *pneumatic bone growth begins as the infant assumes respiratory life.*

On the other hand, the mesially placed *petrous apex* is made up of (2) *actively protective, marrow-filled spongiose bone,* whose (a) biocellular somatic osteogenesis, beginning in the fœtus coincident with the establishing of the blood circulation at about four and one-half months of uterine life, is (b) *continued through infancy and into childhood;* that is, as long as the bone increases in size; after which the bone of the apex becomes senescent as a proliferating growth organ.

Safely placed between the (1) passively protecting tympano-antral mastoid and the (2) proliferating apex is (3) the *special-sense labyrinth capsule,* whose non-vascular compact tissue bone has passed through maturity and become (d) *senescent bone prenatally* (See page 41).

The (1) *Senescent "Neonatal Labyrinth Capsule" of the Newe-born Infant Differentiated from the* (2) *"Matured Capsule of Childhood"* — The bony labyrinth is the *opus magnum* of specialized bone architecture that has been evolved by life. For the ebony-like labyrinth of the infant — for which I offer the designation "neonatal labyrinth capsule" (see page 27) — must be differentiated surgically from the matured capsule of childhood. The latter is formed by the neonatal capsule becoming incompletely surrounded by a "secondary bone shell."

(a) *The Growth of the "Neonatal Labyrinth"* — The neonatal labyrinth is made up of endochondral bone. It springs from the idiocratic cartilage anlage during fœtal life, which in turn came from the otic vesicle of the embryo. Its ossification begins as early as the fourth month of fœtal life (coincident with the "quickening of the child") and continues through the remaining months. It is, thus, (a) full-sized, (b) senescent bone at the time of the birth of the child (Fig. 6) (See page 31).

Thus, not only the (a) pattern of growth of the neonatal labyrinth capsule bone, but also the (b) osteogenic process forming it is unique; for it is the only bone in the body which is (1) full-sized, composed of (2) senescent bone, and (3) is without vessels at birth.

FIG. 6 — (Eagleton) — Infant's labyrinth, full sized at birth, compared with adult bone. Bone grows outward by pneumatic growth of mastoid and inward by marrow growth of apex.

I. Surgical Application of Labyrinthine Infections

Infection Cannot Invade the Neonatal Labyrinth Capsule — The bone of the primitive neonatal labyrinth is impermeable to all types of infection, because of (a) the ivory-like nature of the compact tissue which forms it, and because of (b) the absence of nutrient blood spaces within the bone.

"Matured" Labyrinth Capsule of Childhood — However, outside the labyrinth there grows, during infancy, peripheral periosteal bone, and this is filled with blood vessels. The manner of growth of the shell over the neonatal labyrinth is analogous to the growth of a deer's antlers, that is, by bone growing from a periosteal vascular bed, which forms the springtime fuzz on the deer's antlers.[19] (See page 35).

(b) *The Secondary Ectochondral, Peripherally Formed Periosteal Bone which "Fills in" Areas of the Labyrinth Capsule* — During the first years of postnatal life (a) peripheral growth increments begin to form (b) ectochondral bone around the neonatal labyrinth in definite areas, which continue highly vas-

19. MacEwan, W.: *Growth and Shedding of the Antler of the Deer*, Jour. Anat., vol. 59, 1924-25. (Maclehose, Jackson & Sons, Glasgow, 1920).

cular as long as the "secondary periosteal shell" is growing. Some of these areas continue as cancellous bone throughout life although their nutrient channels are empty of blood after the completion of the bone's growth; however, many of the vascular channels are still pervious to very small particles; as they can be artificially injected by pressure from neighboring venous radicles that empty into the jugular bulb. These empty minute microscopic plexuses, which are especially numerous within the solid angle of the petrosal sinus, I have designated "points of venous vulnerability."[20] For they are potential pathways through which, in inflammatory congestion, infection — especially the encapsulated pneumococcus (see page 30) — ofttimes invades a venous sinus, giving rise to (a) infective thrombophlebitis, or a phlebitic process that may extend inward into the arachnoid mesh, and result in a (b) local or a (c) general leptomeningitis.

II. Surgical Application of Perilabyrinthine Infections

The Influence of Vascularity as Affected by the Maturity of the Bone on the Formation of Caries in Areas Adjacent to the Neonatal Labyrinth — (See page 32) — Thus, the exemption of the compact bone of the neonatal labyrinth from infection does not apply to the perilabyrinthine areas which are formed of secondary appositional bone. For the cancellous bone of the incomplete peripheral shell that develops during the first two years of life may be the seat of a long suppurating sinus. Such a fistula may (a) extend above, (b) behind, or (c) below the labyrinth, and may (d) enter the petrous apex. Consequently, in cases presenting symptoms of apical irritation, the surgeon, during operation, should thoroughly investigate the bone (1) *within* the solid angle of the petrosal sinus, (2) about the eminentia arcuata; as well as in (3) the supratubal region, before perforating into the apex itself. For if the external orifice of a fistula be found, it will furnish a "tract" to that part of the apex which is the site of the suppuration.

Varying Forms of Reaction to Infection from Genetically Different Bone — Each of the three genetically different bones — (a) the pneumatic protective special-sense bone of the mastoid, (b) the unique special-sense labyrinth, and (c) the somatic marrow-filled apex — has a (1) specific histological structure because of its generic origin, and in each area the (2) reaction of the bone to infection is different; which reaction is modified by the stage (3) of growth in which the bone is at the time it is attacked by disease. Thus, both factors, (a) the genetic origin

20. Eagleton, W. P.: *Cerebrospinal Fluid as an Aid to Diagnosis and Treatment*, Jour. Laryngol. & Otol., 44:666-667 (Oct.) 1929.

of the bone, and (b) the stage of its growth, have an influence upon (1) the type of the infective lesion present and (2) the course that the disease will take.

The Pattern of Bone Growth Depends Upon the Plan of Its Blood Supply — The morphological structure of a bone is largely dictated by the pattern of its blood supply. This, in turn, depends upon whether the osteogenetic growth process which is present at the moment is (a) solely vegetative, that is, for osseous proliferation; or is for the (b) differentiation of a specialized function. A combination of (c) vegetative proliferation and functional differentiation is the osteogenetic process, usually active during the expanding periods of skeletal growth; although it is a fundamental law that (d) active growth and specialized differentiation are mutually inhibitory (Boyd).[21]

Pneumatic Growth

(a) *The Osteogenesis of the Pneumatic Protective Special-Sense Mastoid Apophysis; and that of the Olfactory Domain as Furnished by the Pneumatic Portion of the Respiratory Ethmoturbinal Nasal Capsule* — The bones of the (1) mastoid apophysis, and the (2) respiratory portion of the nasal capsule expand by pneumatization. This pneumatic growth occurs during the first general "filling-out cycle" in infancy. But the growth of the skull does not run parallel with the growth in the size of the body, as cranial bone growth is regulated by the demands of its protective nature. For protection (1) from external trauma or (2) against invasion by microörganisms is required as well as an increase in the size of the (a) face, of the (b) neural tissues of the brain and its end-organs; each of which factors comes into play at different periods of fœtal, infantile and childhood life.

Bones that expand by pneumatic bone growth are "surface" bones. The environmental requirements for the growth of a mammalian protective pneumatic bone are — (1) previous ossification in the fœtus from (a) a cartilage anlage, with a layer of (b) "investing" membrane bone covering; the bone spaces between the two strata of genetically different bone being filled with (2) infantile fat at birth. It is the entry into this fat-filled cleft of a (3) rapidly proliferating surface epithelium from the upper respiratory tract that furnishes the stimulus resulting in pneumatic bone growth during infancy and early childhood.

21. Boyd, W.: *Growth, Normal and Abnormal*, J.A.M.A., 105:1520 (Nov. 9) 1935.

(b) *Pneumatic Growth in the Antrotympanic Domain — Small-cell pneumatic growth of the mastoid*, to furnish protection for the labyrinth from external violence, is very active during the first year of infancy. Pneumatic growth then progresses slowly but evenly up to maturity, as a part of the general growth filling-in process. Thus, in the tympano-antral domain, immediately with the assuming of respiratory life, pneumatic growth begins between the bone which sprang from the cartilage anlage of the antrolabyrinth capsule, and its membrane-bone covering. This area in the fœtus had developed from a primordial cranial base cartilage, to which an element of branchial arch cartilage (Reichert's cartilage) and an ectodermal quotient (Stone[5]) had been added. (See pages 25 and 34). Pneumatization occurs by this cleft being invaded by a rapidly proliferating epithelium[22] from the upper respiratory tract. This invading epithelium, while absorbing the primitive mesenchyma (Wharton's jelly) that fills the cavity of the antrotympanum and laying down an epithelial lining, replaces the infantile fat that fills the spaces in the primitive mastoid.

The rapidly proliferating epithelium thus converts the tympano-antral cavity and the primitive mastoid cells into pneumatic spaces, furnishing them with a thin epithelial lining. A multiplication of new pneumatic spaces now becomes the osteogenetic process to complete growth's lateral expansion of the cranial base outward. This growth process of pneumatic bony expansion proceeds very actively during infancy, continuing slowly for about the first three years. During this time air cells frequently extend into the zygomatic and squamous areas of the temporal or even the occipital bone. It is a later extraterritorial pneumatization that invades a petrous apex in about 35 per cent of individuals, but this only happens after the apex has stopped growing, its red marrow having been converted into "acellular fat."

Summary of Pneumatic Growth of the Mastoid Apophysis — Thus, the mastoid apophysis is a neurocranial protective special-sense (vestibulo-auditory) bone. It increases in size by osseous palingenesis (Hæckel); that is, through the successive formation of additional small air-filled spaces (Eagleton)[23] of the parent type. The outer wall of the mastoid apophysis is "ballooned outward," as it were, from the domain of the tympanic antrum by peripherally formed pneumatic bone growth during infancy and childhood.

(c) *The Function and Pattern of Pneumatic Bone Growth* — Mammalian pneumatic bone is a type of passively protective surface special-sense meso-ectodermal archegenesis. Pneumatic bone growth in the mammal is a specialized modification of the

22. Wolf, Dorothy: *The Microscopic Anatomy of the Eustachian Tube*, Ann. Otol., Rhinol., Laryngol., 43:483-494 (June) 1934.

23. Eagleton, W. P.: *Suppurative Meningitis of Otitic and Nasal Origin; Its Relation to Blood Stream Invasion of the Pial Vessels*, Arch. Otolaryng., 15:885-905 (June) 1932.

pattern of bone growth which was evolved by avian life. The function of pneumatized bone in birds is for the storage of oxygen during prolonged flight.[24] In the mammal, pneumatic bone growth is an adaptation of "surface membrane" bone, its specific function being protection of the special-sense end-organ for hearing against violence, and the domain of the special olfactory sense against the infective organisms which are constantly entering the nose.

III. Surgical Application of Pneumatic Bone Infections

(1) *The Limitations of Infection in Coalescing (Kopetzky) Mastoiditis Occur Because of the Pattern of the Blood Supply of the Mastoid* — (See page 30) — The blood vessels that develop during pneumatic "ballooning outward" in the growth of the auditory domain run in the submucosa of the proliferating epithelium that lines the mastoid cells. These vessels, once the size of the apophysis has been completed, have no communication with the deeper adjacent tissues; consequently, an infection which travels by direct continuity of tissue suppuration is apt to remain localized in the mastoid cells, provided that infection occurs after early childhood; that is, after the absorption of the infantile fat in the bone. However, during infancy mastoid infections are osteomyelitic processes because of the presence of fat marrow.

The Reaction of Undifferentiating Mesenchymal Bone to Infection — Senescent pneumatic bone, after completion of growth in early childhood — such as the fully developed mastoid cells — reacts to infection by transudation of cells (leucocytes, erythrocytes), which are brought to it by the circulation.

Surgical Application of Pneumatic Bone Infections (cont'd)

(2) *Limitation of Abscess Formation in Mastoiditis in Relation to Blood Supply* — Mastoiditis of the confluent carious types is apt to be associated with abscess formation, the abscess being limited by a layer of granulation tissue, round cells, and phagocytosing polymorphonuclear leucocytes, which are brought to the mastoid by the hematic circulation.

The Reaction of the Endothelial Content of Bone to Infection — Marrow-containing bone, that is, mesenchymal bone with an endothelial content — such as the sphenoidal basis and a growing petrous apex — reacts by elements (reticulum, histocytes, and wandering macrophages) present in the marrow itself.

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24. Meltzer, S. J., and Meyer, A. L.: *Experimentally Continuous Insufflation Through the Humerus in Fowls*, Am. J. Physiol., 40:126, 1916.

IV. Surgical Considerations in Infections of Marrow-Containing Bone

(1) *Direct Control of Infection* — Thus, the reaction against bacterial infection in marrow-containing bone is a direct local process, which is more efficient than the slower one which occurs in pneumatic bone, because the latter is a part of a systemic reaction. For there is evidence that under certain conditions there exists a truly *localized form of immunity* which is *confined to localized areas* or cell groups of the body. This local or tissue immunity is not a desensitization, but is of the nature of a cell immunization (Gay[25]).

Non-Reaction of Special-Sense Bone — On the other hand, (3) *special-sense* meso-ectodermal compact tissue bone — such as the senescent labyrinth capsule, or the compact bone of the visual or olfactory domains — does not react directly to infection. This is largely because of an absence of both cellular spaces and a blood supply within the bone itself.

The Actively Working Biocellular Red Marrow-Filled Bones of the Cranial Basis — The bones of the craniobasis, formed by the petrous apices, the sphenoidal basis and the segmental portion of the occipital bone surrounding the foramen magnum, all spring in the fœtus from primordial somatic proliferating cartilage; and all contain red bone marrow at birth.

The Modification of Somatic Bone Growth Processes in the Formation of Special-Sense and Protective Special-Sense Bones of the Neurocranium — Thus, taking proliferating growth in cartilage as a model, life has formed a neurocranial bone by modifications of the progressing stages of somatic bone growth; that is, by adding to, omitting from or combining one or more stages differently. (See page 25).

A special-sense bone grows endochondrally from cartilage; the same as occurs in the growth of somatic cartilage bone, but during its growth (1) endothelial permeation does not occur; so that a special-sense bone matures during fœtal life, and at birth is largely senescent bone. It is still a living but not a live bone.

Thus, special-sense bone is formed in fœtal life by (a) the omission of the stage of endothelial infiltration which develops bone marrow in somatic bone; as well as by (b) the omission of the process of absorption of the calcified matrix by peripheral periosteal bone vessels that occurs in the growth of somatic bone

25. Gay, Frederick P.: *Local and Tissue Immunity* in *The Newer Knowledge of Bacteriology and Immunology*, Chicago, University of Chicago Press, 1928, p. 881.

and thus enlarges the marrow spaces in centrally growing endochondral somatic bone.

The Senescent Neonatal Bone Capsule for the Neural Membranous Labyrinth — There is complete absence of protoplasmic infiltration in the growth of the neonatal labyrinth, which at birth is composed of full-grown compact tissue bone. (See page 35).

However, the neonatal labyrinth has on its anterior and posterior surfaces peripherally formed bone, which adds growth increments to those parts of the cortex that are situated next to the dura. Peripheral periosteal bone continues to grow around the neonatal labyrinth during the first two years of the infant's life.

Passively protective pneumatic bones of the (a) mastoid process and the (b) respiratory portion of the nasal capsule increase in size during infancy and early childhood. The primordial bone itself grows in the fœtus by *combining* (2) *endochondral bone* growth (without marrow) with a layer of surface (appositional) bone. Between the two strata at birth is placed (3) infantile fat (see page 27); fat which, in the somatic bones, is the terminal stage of endothelial red bone marrow infiltration in the areas of the neurocranium that are to extend by pneumatic growth, appears before the completion of proliferative osteogenesis.

Thus, the mammal, while in the fœtal stage, prepares for pneumatized bone growth during infancy by substituting a process which is terminal in the growth of somatic bone.

The Passage of Proliferating Growth into a Histogenetic Process for Functional Differentiation as It Occurs Within the Marrow-Containing Bones of the Base of the Skull — The transition from proliferative osteogenesis of growth into histogenetic differentiation for a specific function occurs in the bones which make up the key positions of the cranial basis, because they possess specialized attributes; each attribute being an adaptation and a refinement of a differentiation inherent in all somatic skeletal bones.

These potentialities of the protoplasmic red marrow hemocytopoietic mechanism were gradually augmented in the progressive evolution of the vertebrates; in amphibians, as they passed from an aqueous medium and became the reptilians of terrestrial life.[26]

26. Jeans, James: *Through Space and Time*, London, Cambridge Press, 1934.

The diameter of the base of the mammalian skull expands[27] by the growth of the spheno-occipital basis, in conjunction with the growth of the petrous apices. However, while the morphological pattern of the three bones which compose the framework of the craniobasis is an adaptation of the spongiose bodies of the ring-shaped vertebræ; the manner of growth of an apex is similar to that of the epiphyseal end of the diaphysis of a long bone. The morphological pattern of the infant's craniobasis occurs in a simplified form in all the encircling somatic bones, for the bones of the thorax, pelvis and cerebrospinal axis are active organs for the protection of visceral and neural structures, as well as being supporting skeletal bones. All of them are important units of hemocytogenetic activity, and this continues throughout the entire life of the individual.

The Function of the Marrow-Filled Sphenoidal Basis — The cellular red marrow within the spongiosa of the sphenoidal basis, in addition to its attributes for proliferative osteogenetic growth, acts continuously as the chief biocellular nutritional organ of the cranial basis. It is in this capacity that it becomes the active mechanism which protects the brain's base, especially the pons, against infection.

Thus, surgically, the sphenoidal basis should not be regarded simply as a mechanically protecting bone, but as a constantly working histogenic organ for metabolic activity, possessing specific bactericidal properties; although the matrix of the bone does not increase in size after the ossification of the occipitosphenoidal synchondrosis, which is completed at about the eighteenth year.

The Specific Functional "Differentiation" of the Marrow Within the Sphenoidal Basis — Thus, the sphenoidal basis has (a) a distinctive embryological structure because of its (b) unique position during the growth of the craniobasis and, because, during life, it is (c) exposed to infective organisms from the upper respiratory tract. Its protoplasmic growth potentialities dictate the (1) distinctive pathognomonic character of the infective lesions that may occur in the sphenoidal marrow, and furnish its (2) phenomenal osteogenetic powers of repair. (See page 46).

The Sinusoidal Epithelial Reticulum of the Bone Marrow — The endothelium of the sinusoidal spaces within the reticulum of bone marrow is functionally and morphologically different from the "surface endothelium" that lines the vessels of the blood

27. Eagleton, W. P.: *Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex*, Arch. Otolaryng., 13:386-422 (Mar.) 1931.

circulation; although the sinusoidal spaces empty into the capillaries of the circulatory system. For the character of the endothelial sinusoidal space of the bone marrow changes immediately as it becomes the endothelium of the circulatory capillaries (Minot[28]); which cannot generate blood cells, a differentiation function possessed by the sinusoidal endothelial reticulum.

THE HEMOCYTOGENETIC RETICULUM OF BONE-MARROW; *a Part of the* (A) *Reticulo-Endothelial System of Fixed Tissue Cells (Ashoff and Kizono). It Is Well-Named When It Is Acting in the Development of Bone. The System Is More Appropriately Designated* (B) *Macrophagic (Metchnikoff, and Evans), When It Is Operating Under Stress, Combating Infection and Taking Part in Bone Repair; and* (C) *the Reticulo-Histocytic (Maximow) System When Functioning Metabolically, Which Includes Its Routine Bactericidal Activity.*

(A) Ashoff's reticulo-endothelial system is the generic term for that rudimentary connective tissue network which is scattered so freely throughout the body. But as the morphological character of its elements is constantly changing — depending upon the function that the cells are performing at the particular moment — I would hold that the term "reticulo-endothelium" is descriptive of those morphological features of its osseous units which are outstanding during the normal successive process in growth; at which time the reticulum and sinusoidal epithelium of red marrow play the chief rôle in proliferative osteogenesis.[29]

(B) *The Macrophage System in Local Immunity* — On the other hand, the term macrophage (Evans) system is descriptive of those large mononuclear cells of the marrow which operate actively and are to be found in large numbers when the bone marrow is reacting under the stress of (a) a locally acting infection, or (b) in bone repair. For the large motile mononuclear phagocyte and its progenitor, the fibroblast,* both spring

28. Minot, S. R.: Proc. Boston Soc. Nat. History, 29:185, 1901.

29. Leriche, R., and Policard, A.: *Normal and Pathological Physiology of Bone.* Translated by S. Moore and F. A. Key, St. Louis, C. V. Mosby Company, 1928.

* NOTE: "Monocytes, macrophages and fibroblasts are closely related forms. A macrophage is merely a monocyte (large mononuclear leucocyte) in a more active metabolic condition."
Carrell, A., and Ebeling, A. H.: *Properties of Fibroblasts and Macrophages,* J. Exper. Med., 44:30 (Sept.) 1926.

from the reticulum of the cellular marrow of bone. They are probably the chief elements which combat all foreign irritants that may invade the sphenoid basis. For mononuclear phagocytes gather in the medullary tissue during active bone absorption (Shipley[30]), and during infection of the petrous apex and sphenoidal basis.

Furthermore, it has been experimentally demonstrated that the greater the number of mesenchymal mononuclear cells (macrophages) mobilized within a tissue, the greater is the local cellular immunity against infection (Gay and Morrison, 1923); and that the very act of phagocytosis by histocytes and mononuclear cells performs an important rôle (Clark[31]) in the production of immunity.

Surgical Considerations in Infections of Marrow-Containing Bone (cont'd)

(2) *Local Areas of Immunity from Infection* — Functionally, then, the sphenoidal base reacts locally to infection in a way analogous to that of other areas which possess specific immunity against certain types of infection, viz., the connective tissue of the skin against anthrax (Besredka[32]), the lining of the serous cavities against streptococci (Guy[25]), the mesenchymal lining of the septal alveoli of the lungs against pneumococci (Fried[33]), and an organized group of histocytes in the gall bladder (Cooke[34]).

The Potential Activity of a Petrous Apex in Combating Infection — A similar potentiality is inherent within a petrous apex; which, however, is operative only when its spongiosa contains red marrow. For after its marrow becomes acellular fat, or its bony laminæ are partially converted into air spaces, an apex, to regain active immunity properties, must again become filled with cellular red bone marrow. (See page 40).

30. Shipley, P. G.: *Special Cytology*, New York, Hoeber, 1928, p. 725.
31. Clark, Ada Ramkey: *The Rôle of Clasmatocytes in Protection against Pneumococcus*, Arch. Path., 8:473 (Sept.) 1929.
32. Besredka, A.: *Local Immunization*, Baltimore, Williams & Wilkins Co., 1927.
33. Fried, B. M.: *Defensive and Metabolic Apparatus of the Lungs*, Arch. Path. & Lab. Med., 2:1008 (Dec.) 1926.
— *The Origin of the Histocytes (Macrophages) in Lungs*, Ibid., 3:751 (May) 1927.
— *The Lungs and the Macrophagic System*, Ibid., 17:76 (Jan.) 1934.
34. Cooke, H. H.: *The Reaction to Foreign Material in the Normal and in the Inflamed Gall Bladder*, Arch. Path. & Lab. Med., 14:856 (Dec.) 1932.

SURGICAL CONSIDERATIONS (cont'd)

(3) *Limitations of Local Protection by Granulation Tissue* — For it has been established experimentally that while granulation tissue usually resists infection from streptococci through round-cell infiltration, in certain areas (such as the pleura) polymorphonuclear leucocytes fail to furnish protection; however, the histocytes and macrophages — the large mononuclear group of cells of the mammal — do furnish immunity to such tissue that normally contains them (Gay and Clark[35]).

(C) *The Reticulohistocytic System* — Finally, the term reticulohistocytic system (Maximow) well describes the bone marrow mechanism while it is carrying on its normal metabolic functions, a part of which is phagocytic, in keeping the circulating blood stream purified.

The Specific Function of Histogenetic Differentiation Against Infection Possessed by the Sphenoidal Basis, to which the Petrous Apices Are Allied — The bony spheno-occipital basis is an adaptation of the morphological structure of the bodies of the vertebræ, the spongiose cavities of which continue filled with red marrow throughout life.

The proliferating somatic cartilage from which springs the sphenoidal basis — the pre-vertebral, improperly named, segment of Kolliker — is converted into bone early during fœtal life. Its red bone marrow constitutes the chief osteogenetic agent for proliferative growth of the base of the skull; in which capacity it acts continuously from the time of the establishment of the cerebral blood circulation during the fourth month of fœtal life.

But after birth, the sphenoidal red bone marrow assumes, in addition, a metabolic function of a specific character; acting now routinely for the local protection against infective agents that may enter by the oropharynx of the meningeal mesh which surrounds the base of the brain.

This osteohemogenetic mechanism (to prevent infective agents that may be in the oropharynx from reaching the meninges) is developed during the growth of the infant. For it is at this time that the burden of protection of the upper respiratory tract through a process of local immunity is gradually transferred from the primitive lymphoid system in the neck[36] and throat (with its chief

35. Gay, F. P., and Clark, A. R.: *Further Note on Relative Protection by Polynuclear and Mononuclear Cells in Local Streptococcus Infection.* Proc. Soc. Exper. Biol. & Med., 27:995 (June) 1930.

36. Kingsbury, D. F.: *The Development of the Human Pharynx; The Pharyngeal Derivatives,* Am. J. Anat., 18:329-397, 1915.

visceral organ, the spleen), to the circulatory hemic system with its reticulo-endothelial units in the bone marrow, with its main purifying organ, the liver; the spleen having become the organ of cell storage and cell death in the hemocytopoietic equilibrium (Koumbhaar[37]). Thus after the nursing period of life the red bone marrow of the sphenoidal bases becomes the chief active mechanism of protection to the meso-ectodermal tissues covering the brain.

Surgical Application of Infection of Marrow-Containing Bone (cont'd)

(4) *No Lymphatic Enlargement in Osteomyelitis after Infancy* — After early childhood, infections within the nasal sinuses (an osteomyelitis of a bone of the skull) are associated with but slight lymphatic enlargement.

The Calcified Matrix and the Marrow of the Petrous Apex Spring, in the Embryo, from Proliferating Cartilage. The Growth Process is Vegetative Osteogenesis for the Lateral Expansion of the Cranial Basis. Vegetative Osteogenesis of the Apices is Completed during the General "Filling-out" Cycle in Childhood Coincident with the Massive Pneumatization that Begets the Sphenoid Sinus — The contour that an apex will assume is dictated long before birth. Its calcified matrix and substantia spongiosa both spring from the laterally placed parachordal cartilage, which, unlike the meso-ectodermal neurocartilages from which the protective mastoid and the special-sense labyrinth are developed, is probably of pure mesodermal origin. The conversion of senescent cartilage into bone during fœtal life is associated with the intrusion of protoplasmic hemocytopoietic red bone marrow; the same as is the process of growth in all somatic spongiose bone.[38]

Proliferative Osteogenesis of the Petrous Apices — Likewise, the proliferative osteogenesis of a petrous apex is due chiefly to the biocellular growth mechanism contained within the red bone marrow of its spongiosa. For the petrous apex, at birth, is filled with red bone marrow,[39,40] which continues as long as its osseous structure is increasing in size, after which the apex

37. Koumbhaar, E. B.: *Functions of the Spleen*, Physiol. Rev., 6:160, 1926.
38. Harris, H. A.: *Bone Growth in Health and Disease*, Oxford University Press, London, Humphrey Milford, 1933, p. 248.
39. Cunningham's *Textbook of Anatomy*, Ed. 5, London, 1921, p. 290.
40. Van Noorden, W.: *Contribution to the Anatomy of the Cartilaginous Base of the Skull in Human Embryos*, Arch. f. Anat. u. Entwicklungsgeschichte, Anatom. Abtlg., 1887, p. 241.

becomes senescent bone, containing fat, or becomes either partially or completely pneumatized.

Hemocytopoietic Growth in the Petrous Apex — Thus, during growth, the morphological pattern of an apex is a modification of the active red marrow-filled functioning organ of all spongiose bone, although its mode of growth is that of the epiphyseal end of a metaphysis. For the apices, during infancy and early childhood, are among the marrow-containing, dynamic growth organs that form the mesial portion of the central domain of the cranial basis.

FIG. 7 — (Eagleton) — Hyperplastic red bone marrow filling sinusoidal bone spaces in apex in attempt to control infection.

V. Surgical Application of Apical Infections

(1) *Anatomical Types of Osteomyelitic Infections of the Apex during its Proliferating Stage*—Thus, during the period of lateral expansion of the cranial cavity, a petrous apex has the inherent character of both the spongiose body of a vertebra and of the epiphyseal end—the growing end—of a metaphysis. Consequently, it may be the site of forms of osteomyelitic infection that occur in either (a) the body of a vertebra, or (b) in an epiphysis of a growing bone. But because of its fixed position the petrous apex is largely immune to those types of disease that flower under movement, such as tuberculosis and cancer, frequently present in a vertebra or a tubular bone.

The Fat Marrow of the Apex, a Senescent Protoplasmic Process which may be Followed by an Overflow Pneumatization during the "Filling-out" Period of the Skull's Growth—Having fulfilled its proliferative function in the growth of the bases, an apex becomes a senescent organ, its red marrow turning into acellular fat; after which the apex may be invaded by an overflow pneumatic process, in which both its fat marrow and bony trabeculæ may be absorbed. (See page 26).

Surgical Application

(2) *Types of Lesions in Apex*—As long as apex is fat filled, any infection within it partakes of the properties of an osteomyelitis; but when an apex is fully pneumatized, an apical inflammatory process may assume any of the (a) various types of osteites so familiar in mastoiditis. However, if there still be any fat remaining, any infection within an apex is apt to be (b) modified by the primitive marrow-containing nature of the bone. (See Fig. 7).

V. *ACUTE SEPTIC OSTEOMYELITIS OF THE PETROUS APEX (ACCOMPANIED BY A POSITIVE BLOOD CULTURE), AND ITS ASSOCIATION WITH MENINGITIS FROM THE INFECTION PASSING THROUGH THE DURA.**

The osteomyelitis may be (a) hemorrhagic, or (b) phlegmonous, or (c) gangrenous in form; (a) "fulminating," (b) invasive, (c) "resting," or (d) "recrudescent" in character; (a) diffuse, or (b) local in extent; and (a) embolic in origin, or due to (b) an extension of a thrombophlebitis from an adjacent area.

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* Note: *Clinical Types of Apical Suppuration and Associated Osteomyelitic Lesions of the Skull in Relation to Meningitis* (Based on 42 cases of Apical Suppuration and 24 cases of Sphenoidal Bone Disease Associated with Meningitis). Parts V to IX of classification submitted to Amer. Otol. Society at Toronto, Canada, on May 28, 1935.

(1) Acute fulminating and invasive osteomyelitis; "acute diffuse hematogenous osteomyelitis" of the petrous apex — a bone marrow sinusoidal-space infection.

(2) Localized hematogenous osteomyelitis.

(3) "Subacute thrombotic (a) localized or (b) diffuse osteomyelitis." It may suddenly transmute into

(4) "A fulminating osteomyelitic phlegmon with an associated phlebitic meningitis," either from operative trauma or upon the advent of a concomitant infection, especially an angina.

VI. *"Embolic diffuse hematogenous osteomyelitis."* It may be limited to (1) one apex, with an associated (a) embolic mastoiditis and (b) meningeal irritation; or it may involve (2) both apices, because the infection is in the blood sinusoidal spaces of the marrow.

The embolic character of the infection is suggested by (a) early spontaneous rupture of the drum membrane; its (b) meningeal character, by (c) an intermeatal type of facial weakness, or by (d) pain behind either eye; the (e) exudate within the pontine cistern can be diagnosed by the absence of, or a great reduction in, the nystagmus induced by turning.

Shortly after invasion of an apex by an embolic hematogenous osteomyelitis, the process is apt to become (a) semi-acute, from which it passes into (b) a chronic bone septic stage, or becomes (c) resting, and results in (d) resolution. However, it may (e) recrudesce, especially if the infected blood channels are traumatized by operation, or the infection is transmitted by an anginal inflammation.

VII. *"Recrudescent (thrombotic) osteomyelitis* associated with an immediate and continuous blood infection (positive blood culture) and with a thrombotic meningitis or meningo-encephalitis."

VIII. *ASSOCIATED OSTEOMYELITIC INFECTION OF THE MARROW-FILLED BONES OF THE CRANIAL BASE.*

"Sinusoidal-space osteomyelitic phlegmon of the sphenoidal basis" causing diffuse embolic foci, with terminal subdural-space meningitis.

IX. *"Chronic necrosing osteomyelitis of the sphenoidal basis,"* which, if traumatized by operation, may cause a fulminating meningitis.

The Potential Protective Properties of the Apex upon the Approach of Infection — As long as an apex contains acellular fat, it has the potentiality of metaplasia, that is, of being transformed, under the stimulation of an approaching infection, into its original red marrow state (Custer[41]); in which case, the apex again becomes an active biocellular mechanism against infection.

41. Custer, R. P.: *Studies on the Structure and Function of Bone Marrow. I. Variability of the Hematopoietic Pattern and Consideration of Method of Examination,* J. Lab. & Clin. Med., 17:951 (July) 1932.

a property it possesses in common with all primordial red marrow-filled bones.[42]

Summary of Functional and Structural Potentialities Connected with the Growth of the Petrous Apex—(1) Primarily the petrous apex is made up of (a) a mesodermal bony framework having (b) an endothelial content, (c) modified by its proximity to meso-ectodermal tissues, viz., the special-sense neurocranial labyrinth and the brain's dural envelope. (2) The endothelial bone marrow within the osseous apex, after serving (a) its vegetative purpose for growth during childhood, (b) becomes senescent; (c) the functioning red marrow being converted into fat. (d) Until acellular fat has replaced cellular red marrow, apical spaces cannot be pneumatized; while an infective inflammation within an apex will partake of the nature of an osteomyelitis.

However, all through life, any (3) fat marrow that may remain within an apex retains the potentiality of again becoming cellular red bone marrow, under stimulation of infection; which will enable it to regain its original specific bactericidal properties and exceptional reparative powers.

Surgical Application of Apical Infections (cont'd)

(3) *Frequency of Spontaneous Cure of Apical Infections*—It is because of this potentiality that a local abscess is so infrequent during an apical infection; although a homolateral mastoid suppuration is often complicated by a reparative osteomyelitic apicitis. For fully 80 per cent of all apical infections are cured simply by drainage of the causative mastoid suppuration.

(4) *The Unique Type of Blood Supply of the Bones Forming the Pre- and Para-chordal Areas of the Cranial Basis Makes the Formation of Sequestra Infrequent in Suppurative Lesions Confined to a Petrous Apex or to the Sphenoidal Basis*—The sphenoidal basis and, to a much lesser degree, the petrous apices have adequate blood supply from their own sinusoidal spaces, and, in addition, receive blood from independent vascular systems. It is this vascularity that prevents the formation of bony sequestra in pyogenic lesions limited to a petrous apex or the sphenoidal basis; in contradistinction to the large sequestrum which forms so quickly and is so frequent in osteomyelitis of a long bone, following an occlusion of its central nutrient "end" vessel.

Likewise, in osteomyelitis of the calvarium numerous small sequestra are common although they form slowly, because a vault bone possesses a central nutrient blood supply inadequate to continue the nutrition of

42. Mueller, E. J.: *Knochenmark und Leukocyten Beiträge zur Pathologischen Anatomie der Leukocytose*, Virchows Arch. f. path. anat., 246:49-88, 1923.

its osseous framework when (a) its own nutrient radicles, or (b) an associated vessel coming from the surrounding soft parts or from the dura is attacked by an advancing thrombosing infective process.

The Relation of Infantile Fat Bone Marrow to Pneumatic Growth — As previously stated, at birth all the bones of the skeleton have sprung from cartilage and contain red bone marrow in which there is no microscopic fat (Piney[43]); excepting the (a) special-sense and (b) the passively protective bones of the skull. Infantile fat, however, fills the bone spaces in the primitive mastoid and in the respiratory portion of the ethmoid labyrinth; both of which areas extend by small-cell pneumatization during infancy.

The bone of all protective pneumatic special-sense domains is formed in the embryo by the combination of two osteogenetic processes in a manner distinctive for the mammalian cranium. For at birth a mastoid or respiratory nasal bone, although it sprang in the fœtus from a cartilage anlage — and is thus (1) intercartilaginous bone — is covered by (2) a layer of appositional membrane bone, with (3) infantile fat filling in the bony spaces (4) between cartilaginous and appositional bone strata. (See page 26).

"Extraterritorial Pneumatization" is a Secondary Growth Process — Other bones in domains which are not the intrinsic sites of pneumatic growth, such as the petrous, may become air-filled, but pneumatization can occur only after the skull's internal capacity is completed, and not during the primal formative growth period of the base. For extraterritorial pneumatization is possible only when the regional histogenesis of the bone growth has run its normal course, and the red bone marrow of the apex has become converted into fat, that is, the osseous proliferative function — osteogenesis for growth — has passed, and the bone become senescent as a growth organ.

Pneumatization of Apex

(1) *A pneumatic overflow from the mastoid invades the petrous apex in about 35 per cent of Caucasians* (Glick[44]).

(2) *Pneumatization of a petrous apex is an "overflow replacement," not a bone expanding process.* "Extraterritorial pneumatization" is an overflow replacement, not an expansion growth process. It does not increase the capacity of the skull or the size of its bones.

43. Piney, A.: *The Anatomy of the Bone Marrow,* Brit. M. J., 2:793-794 (Oct. 28) 1922.
44. Glick, N. N.: *Microscopic Observation of the Petrous Apex,* Ann. Otol. Rhinol. & Laryngol., 42:175 (Mar.) 1933.

In the mammalian cranium the object of overflow pneumatization is to equalize inequalities in its external and internal surface that may have appeared during the growth of the skull.

"Overflow pneumatization" has a different functional purpose from that of pneumatized growth, although it is a related growth process. For overflow pneumatization occurs at a later period in the growth of the skull, i.e., in childhood, after the size of the apex has been fully determined, and the end-organs of hearing and equilibrium have been furnished adequate protection through the completion of lateral expansion of the skull's base.

Air-Filled Cells Within a Petrous Apex Do Not Increase the Size of the Bone—The filling by air of marrow spaces within an apex does not increase the size nor alter the external shape of the bone, air spaces lined by the most delicate epithelium being substituted for the marrow spaces. This is in contrast to the osteogenesis of the mastoid by pneumatic growth, where the cortex of the bone is "ballooned" outward, as it were, from the mastoid tympano-antral level.

Surgical Application of Apical Infections (cont'd)

(5) *The Influence of Infection on a Partially Pneumatized Apex*—Wherever pneumatization extends into an apex, the area pneumatized loses the differentiation potentialities which are inherent within protoplasmic bone marrow. The bone's protective response now is no longer a locally protective reaction performed by the motile histocytes springing from the endothelial sinusoidal reticulum of the red bone marrow itself, for if a completely pneumatized petrous apex be attacked by infection, its protective reaction is through leucocytes and other elements supplied from the general blood circulation. However, when a partially pneumatized apex becomes the site of infection, any bone marrow which lies adjacent to the infected area may completely control the infection within the infected pneumatized spaces, provided the virulence of the infecting organism is not overpowering; that is, surgically speaking, provided the infective process has a tendency to localize and is not actively invasive.

Plasma-Cell Mastoiditis Secondary to Apical Infection—Furthermore, a low grade infection in an apex, which is connected by air-filled spaces with the mastoid, may awaken a metaplastic process in the marrow, which may extend externally from the infected apex and result in the filling of the mastoid with plasma cells. An avirulent suppuration of an apex thus gives rise to a specific reparative type of plasma-cell mastoiditis.[45]

45. Discussion in Vail, H. H.: *Vidian Neuralgia, with Special Reference to Eye and Orbital Pain in Suppuration of Petrous Apex,* case 2 (W. H.), Trans. Am. Otol. Soc., 22:119, 1932.

SUMMARY:

Pneumatization of a Petrous Apex is a Different Physiological Process from Pneumatic Growth of the Mastoid, and Occurs at a Different Stage in the Development of the Cranial Base. Its Surgical Importance in Infection — Thus, (1) both (a) the period of life during which the spaces in an apex may become air-filled, and (b) the purpose that pneumatization serves, differ; although the method by which air-filled spaces are substituted for bone marrow is similar, in that (2) (a) fat marrow is converted into pneumatic cells by (b) the invasion of rapidly proliferating epithelium.

(3) Fat marrow in an apex, by a process of metaplasia, may again become red marrow and directly control infection.

(4) "Plasma-cell mastoiditis" represents a type of reactive protoplasmic protective process which extends into the mastoid cells from an adjacent area that originally contained red bone marrow, be it an apex or an occipital bone.

(5) *Pneumatization from the apex never invades the sphenoidal basis* even under the hyperplastic pneumatization of acromegaly. For the red bone marrow is never converted into acellular fat marrow; the sphenoidal basis continues throughout life to be an actively functioning hematogenetic organ with inherent bactericidal potentialities, its red bone marrow (a) reacting locally to infection by a protoplasmic growth process, and possessed of (b) specific properties for repair.

VI. Surgical Considerations in Infections of the Sphenoidal Basis and Apex

Consequently, pyogenic lesions of the sphenoidal basis, because of the bone's (a) selective regional position and (b) continuous protoplasmic differentiation, are (1) distinctive. Furthermore, an infective disease of the sphenoidal sinusoidal spaces runs (2) a different course than does infection of the air spaces of the mastoid, or even an infection within a petrous apex.

(3) However, infective lesions of an apex may partake of some of the specific osteomyelitic features seen in pyogenic lesions of the sphenoidal basis, or may possess the characteristics of one of the various types of infections of pneumatic bone so familiar in mastoiditis.

(4) A coalescing type of petrous apicitis can occur only when the apex has been invaded by an overflow pneumatizing process during the growth of the skull's base.

VII. Surgical Considerations in Special-Sense Bones in General

(1) *Passively Protective, Compact (Ebonized) Tissue, Special-Sense Bones Never Contain Bone Marrow*—The special-sense bones (the labyrinth capsule, the perforated plate of the nasal capsule, and the mesial area of the orbital roof) never contain any marrow. The osseous growth of a special-sense bone occurs in the fœtus (a) from cartilage, having (b) membranous (appositional), peripherally formed bone growth increments. Near the median-line area of the anterior two-thirds of the cranial basis, growth increments from the dura form the internal "investing" layer of the calvarium. Thus, the median domain is composed of both dura and bone, which here become one structure that cannot be surgically divided.

(2) *The Liquefaction and the Absorption of the Compact Special-Sense Bones that Follow Infection Are Not the Result of Osseous Reactions*—Special-sense bone, being an inanimate non-vascular osseous structure (the function of which is the passive protection of an end-organ), offers no site in which bacteria can develop or multiply, largely because of lack of protein elements for bacterial food.

The liquefaction and necrosis of compact bones are not due to the growth of infecting bacteria within the bone itself, but are the result of nutritional death following the infecting of surrounding tissues; which later furnish the nutritional juices on which both compact tissue and senescent bone live.

(3) *Absorption of Dead Bone in General*—Dead bone is removed by the phagocytosis of osteoclasts and mononuclear phagocytes in much the same way that bacteria are digested by means of protoplasmic endothelial elements within the bone marrow; for the endothelial spaces of the reticulum are its functioning part. In it originate the large motile mononuclear cells, which not only are phagocytic, but, in addition, are the principal *factor* in local immunity. Thus, the degree of immunity against infection, or the protoplasmic resistance of individual tissue, is dependent upon the number of mesenchymal mononuclear cells present; for the metabolic action of the red bone marrow is rendered possible by the sinusoidal reticulum within the spongiosa of the sphenoidal basis. It is the continuous protoplasmic osteogenesis which enables the marrow content to be constantly functioning, giving to the bone its unique reparative and protective properties. For it is my contention that the sphenoidal basis, all through life, continues to possess the nutritional circulatory potentialities present in many of the other marrow-filled bones only during the period of their expansive growth.

Summary of Embryological Factors and Histological Growth Potentialities Which (1) *Dictate the Form and* (2) *Influence the Course of Infective Lesions Within a Petrous Apex, or of the Sphenoidal Basis*—There are (a) embryological and (b) growth factors, as well as (c) histological peculiarities of structure in the bones forming the mesial area of the cranial basis,

that dictate (a) the form that suppurative lesions assume, and (b) the course that they take.

These embryological and growth factors are:

(1) Functionally, the petrous apices resemble the epiphyseal end of the metaphysis of a long bone during its growing period; while the sphenoidal basis is a modification of the spongiose body of a spinal vertebra. (2) Both the nutrient supply of the apices and that of the sprenoidal basis are of the "anastomosing type"; it is not furnished by "end" arteries; and (3) this vascular pattern continues throughout life. Furthermore, the apices and sphenoidal basis are (4) primarily red marrow-containing bones; (a) the active potentialities of which are preserved by the sphenoidal basis, but (b) are lost by the petrous apices during childhood, after which the cellular marrow of the apex (a) becomes fat or may (b) be replaced by air-filled spaces. The latter occurs in about 35 per cent of all apices. However, because of its primitive hemocytogenetic character, those portions of an apex which still contain fat, may, on the approach of infection, undergo (5) a rapid metaplastic change; their acellular fat again becoming red bone marrow. Thus, a threatened or invaded petrous apex may regain the bactericidal and reparative potentialities which are inherent within the protoplasmic red bone marrow.

CONCLUSIONS

(1) *Protoplasmic Osteogenetic Differentiation Within the Sphenoidal Basis and the Petrous Apex Dictates their Response to Infection* — It is the continuation through life of augmented nutritional properties of growth, largely because of their unique blood supplies from bilaterally placed and freely anastomosing vessels, that gives rise to the specialized marrow-containing bones of the craniobasis — the sphenoidal basis, and, to a lesser degree, the petrous apices — their (a) distinctive reparative activity, as well as (b) their specific biochemical resistance against infection. For these structures have (1) comparative freedom from infection, and (2) specific powers of bone repair; and (3) there is a distinctive character to lesions when they are attacked by suppuration. (See page 34).

(2) *Physiologically Directed Surgery Necessary in Sinusoidal Osteomyelitis* — Surgery can cure a considerable proportion of patients with meningitis, but it must be properly directed surgery. For surgery on an inflamed vessel in the bone, which vessel passes from the bone into the dura, either does good or harm. There is no "harmless exploration" in suppurative disease of the base of the skull. Thus, the problem of meningitis is a *problem of physiology as modified by infection.*

Practical Lessons — This study of bone embryology and of growth has taught me many things. It has taught me how to recognize (1) the dangerous cases of ear suppuration; (2) the cases of apical infection requiring operation, and those in which there should be no surgical trauma; why (3) *Streptococcus mucosus* (pneumococci type III) infection within the ear in an elderly person very frequently causes an explosive terminal meningitis, often after the ear has ceased discharging; while (4) infection from *Streptococcus mucosus* in a child causes mastoiditis, or at most, sinus thrombosis, which an operation will cure. This study explained many things: It explained (5) the "suspicion of facial palsy which is absolutely diagnostic of an advancing lesion within a petrous apex, although no other symptoms whatsoever of

apical infection may be present."[46] It explained why (6) the turning test is a means to diagnose an infection localized in the basal system, and why this occurs before alarming signs of meningeal irritation have become evident.[47] But the turning of a patient in whom disease is situated within small vessels running from the apex into the brain is dangerous, because the pial vessels in a blood vessel infection are apt to be softened; in which case turning may upset that delicate balance which exists between the circulatory and the cerebrospinal systems, and thus a meningoencephalitis, which is beyond the control of surgery, will be precipitated.

The Future of the Surgical Treatment of Otitic and Nasal Meningitis — Chiefly, this study has taught me how little is really known of suppuration of the bones of the base of the skull; and that the treatment of otitic and nasal meningitis today is in its infancy; but like a baby born of healthy and intelligent forebears, it has a golden future perhaps — perhaps just around the corner.

46. Eagleton, W. P.: *Practical Aids to Diagnosis and in Surgical Management of Meningitis from the Ear or Nose*, J. M. Soc. New Jersey, 29:924-932 (Dec.) 1932.
47. Ref. 16, illustration on page 890.

ADDRESS OF THE GUEST OF HONOR

* * *

BRAIN TUMORS AND THE EAR

DOZENT DR. HANS BRUNNER

VIENNA, AUSTRIA

I EXPERIENCED both a happy and proud moment when I received an invitation from your honored president, Dr. Eagleton, to speak before this group. I wish, therefore, to express my most sincere thanks to you and Dr. Eagleton for making such a happy and proud experience possible. Of course, this happiness and this pride did not last very long, because very quickly my worries began when I realized I must talk upon a subject which might justify for me a trip from Vienna to Cincinnati. I must confess that in spite of deep thought upon the problem I could find no subject of that kind. But since I was most interested in coming to America, I have prepared the subject "Brain Tumors and the Ear."

Only one with very original thoughts could have the courage to talk about the essential ear, nose, and throat diseases in such a way that it would be worth while to listen. In that specialty some few men of ingenious spirit have set up the fundamentals of our knowledge. Therefore, in that field a commentary would be superfluous and a critique dangerous. It is much easier when one goes into the borderland of our specialty, especially into a borderland in which neurologists, surgeons, internists, ophthalmologists and otologists do their best to confuse one another. In such a discussion, one may push up waves so high that one may also lose one's self under the waves. This would be especially easy for me, because I have learned in the past few days what seasickness means. Therefore, it makes no difference to me if I must talk about a subject upon which one can not touch without getting "seasick" again. In spite of this, gentlemen, I thank you for accepting the subject which I pro-

posed. You have in that way simplified my obligation very much, because it is much easier to talk about the subject "Brain Tumors and the Ear" than to listen to it.

Although I speak in a country in which Eagleton, L. Fisher, Wilson, Jones, Crowe and others have given important contributions to my subject, it is not my intention to tell you of the different opinions of the various authors. I fear my permission to stay in America would not be sufficient for that. But I ask you for your agreement to refer to my own examinations. Of course, I wish to emphasize that I do this not because I do not know or do not appreciate the work of the authors, but because I wish to simplify my subject as much as possible.

My paper is based upon 117 cases of brain tumors, or of brain diseases with increased brain pressure, which were examined by my assistants and myself between 1929 and 1933. These cases have been patients either of the clinic of Professor Pötzl or of the department of Professor Alexander. In this paper, I wish to express my sincere thanks to Professor Pötzl, who has always aided my examinations with kind helpfulness and interest. It is, of course, my natural wish to remember also with respect and thanks my deceased teacher and chief, Professor Alexander.

The number of cases examined since 1920 is, of course, much greater, but I have chosen just the designated period of time because during this period all cases have been examined by the same technique. I have also chosen only those cases in which the diagnosis has been verified by positive findings at operation or at autopsy, since experience has shown us that a negative finding at operation does not exclude with certainty the presence of a tumor. Finally, tumors of the hypophysis and its surroundings have been excluded because in those cases the otological aspect is, as a rule, not of importance.

Technique of Examination

After an examination of the drum, we have made functional tests of the cochlea in the usual manner. Audiometer examinations could not be done, as we have not had at our disposal the necessary apparatus. After an examination of the cochlea we look for spontaneous labyrinthine symptoms (dizziness, nystagmus, disturbances of equilibrium, and past-pointing). Then we do the bilateral calorization test and finally the caloric test.

When it is impossible to draw conclusions from those tests, we make the usual turning test and occasionally the galvanic test.

For bilateral calorization, we use the apparatus of Ruttin, which I have modified in such a manner that the ear speculæ may be moved in all directions. With the light of the mirror, we bring both speculæ in the external canals so the tips of the speculæ are directed against the drums. This procedure must be made very carefully as results will be in error unless both speculæ lie in the same relative positions in the external canals. Therefore, it is better to check the position of the speculæ before irrigation is attempted. Now, with the head of the patient in normal position, we irrigate slowly with 120 cc. of water (13-15 degrees centigrade) in both ears. The result is positive when horizontal and rotary nystagmus (II degree) appears in from 3-5 seconds after the end of the irrigation. Dizziness without nystagmus, nystagmus (I degree) to one side, or vertical nystagmus are not positive results. The test can be performed in the reclining or sitting positions.

The unilateral caloric test we perform with about 5 cc. of water (13-15 degrees centigrade), which we irrigate with moderate pressure against the drum in normal position of the head or in the optimal position of Bruennings. We consider the result normal when after a latent period of from 15 to 30 seconds a nystagmus of I (or II) degree appears, which lasts about 60 to 90 seconds. This is average time from the experience of many thousands of examinations.

From the unilateral caloric test, we may make a diagnosis of normal irritability, of hypoirritability, of non-irritability, and of hyperirritability of the labyrinth. Although it is relatively easy to diagnose a hypofunction of labyrinth, it takes a much greater experience to diagnose a hyperirritability of slight degree. It is necessary, therefore, to have some outline for that diagnosis. Of course, we must concede that very often in such cases the personal experience of the otologist is of decided importance. Generally, we diagnose hyperirritability when with our caloric test, which we have described above, a nystagmus of III degree appears or when the nystagmus of I degree definitely lasts longer than two minutes. Of course, we presuppose the middle and inner ear to be normal. We do not care very much today about the shortening of the latent period, which was earlier considered

of importance to us. Also, we do not worry very much about the dizziness or the disturbances of equilibrium.

There is no doubt that the method which I have described will appear very primitive to you. However, will you please keep this in mind? When we wish to compare the cinical value of otological examination to the clinical value of the eye examination, it is absolutely necessary to simplify otological methods and to shorten their duration. Otherwise, it is possible that the many very interesting ameliorations of the functional test recommended in recent years change the auxiliary clinical examination of the ear in a physiological experiment from which nobody has any advantage, neither the patient nor the neurologist, maybe sometimes only the otologist who makes the experiment. Finally, we must say that the clinical value of a functional test is not dependent on theory, it is in first line dependent upon the results, and, therefore, you will allow me to tell you something about results with the described methods.

When we, as otologists, wish to examine brain tumors in the right way, it is first of all necessary to answer the question, how will the function of the ear be influenced by chronic increased brain pressure? This question was first studied by Dr. Eagleton in 1912; later, my teacher, Professor Alexander, contributed some important anatomical facts to that question. In spite of all these important contributions, it is necessary to study this question again from the anatomical and clinical standpoints in order to get a reasonably certain basis for our clinical work. It would be, of course, too much to discuss the whole otological symptomatology in the mentioned conditions. We must restrict the discussion to three symptoms only: (1) the acuity of hearing, (2) the caloric test, and (3) the bilateral calorization.

You know very well that we have for a long time differentiated between generalized and localized brain pressure. This differentiation is right even today. *Generalized brain pressure* is the pressure which presses from all directions in nearly equal degree and which is produced by an increase of pressure in the cerebrospinal fluid, or, as we believe today, by an actual swelling of brain. (*Hirnschwelling of Reichhardt.*) *Localized brain pressure* is produced by a condition which consumes space on the inside of the skull, and which is localized to only one area. Localized brain pressure will be, therefore, mostly pro-

duced by a brain tumor. This division of chronic brain pressure is justified less by clinical experience than by experimental observation, because clinically generalized and localized pressure are very often combined. Anyway, we know on one hand of cases which have brain tumors, therefore, localized brain pressure but clinically no generalized pressure; and, on the other hand, we know of cases which have a very marked generalized pressure but no localized pressure, therefore, no tumor. For these reasons we want to see today how generalized and localized brain pressure influence the function of the ear.

A. Influence of Chronic Generalized Brain Pressure on the Function of the Ear

The best known symptom of generalized brain pressure is choked disc. The question arises: Do we also know a choked labyrinth? To answer this question we must examine the influences of generalized brain pressure (a) on the peripheral ear organ, and (b) on the centres of the eighth nerve.

(a) Generalized Brain Pressure and Peripheral Ear Organ —I have examined microscopically twelve temporal bones from six cases in which no tumors of the posterior fossa were present in order to prove that the inner ear was not influenced by localized pressure, but could be influenced by generalized pressure. Two of those cases have been operated upon, four cases died without operation.

Table I

CASE	DIAGNOSIS	Post-operative time	MICROSCOPIC FINDINGS
(I)	Acute œdema of brain		*No changes*
(II)	Hydrocephalus		*No changes*
(III)	Tumor of thalamus		*No changes*
(IV)	Tumor in the left occipital lobe	Death during operation	Small hemorrhages in the modiolus (?)
(V)	Fronto-temporo-occipital glioma, left	17 days	Hemorrhages in right inner ear. Nerve atrophy in basilar coil (?)
(VI)	Very big glioma in the right (and left) cerebral hemisphere	14 days	Petechial hemorrhages in left inner ear. Œdema of D. endolymphaticus on both sides.

In the first place, we see that in the first four cases, which died without operation (only case IV died during operation), the inner ear was completely or almost completely normal, although all cases (with the exception of case IV) showed a marked choked disc. The petechial hemorrhages in case IV are agonal hemorrhages. The hemorrhages are more marked in cases V and VI, which died after operation. We are forced to explain these hemorrhages by the acute changes in pressure, or better said, by the intensive disturbance in the blood circulation of the brain produced by decompression.

We can therefore say: *Our findings show that chronic generalized brain pressure does not produce any marked microscopical changes of the inner ear. But acute variations of brain pressure, for instance, after decompression, can produce passive hyperaemia, stagnation hemorrhages, sometimes, also, serous transudates in the inner ear.* From this it follows that we should not expect in chronic generalized brain pressure any symptoms from the inner ear, with the exception of some slight symptoms from the cochlea and the labyrinth, which appear at times after decompression, but which disappear again very soon.

Real consequences of generalized brain pressure are the so-called *hernias of the brain*, which are often found in the temporal bone. These hernias are seen much more often in the middle fossa than in the posterior fossa, because the middle fossa is, when the body is in normal position, the deepest point of the base of the skull. In the middle fossa, these hernias are situated sometimes on the involved side, sometimes on the healthy side in the roof of the eustachian tube, in the sphenopetrous fissure, and in the big wing of the sphenoid, finally, in the roof of the middle ear in its anterior part. These hernias can get so large that they may perforate bone and push forward the mucous membrane of the middle ear without perforating it. I only wish to mention the importance of these hernias for the conduction of inflammations from the middle ear to the brain. It is quite interesting that we also find these hernias in the same places without generalized brain pressure. We refer to them as "physiological hernias" and explain them on the basis of transitory variation in generalized brain pressure, which happens in all our lives.

(b) Generalized brain pressure and centres of the eighth nerve — Just as poor as the microscopical findings in the inner

ear are the findings in the brainstem in chronic generalized brain pressure. There have been found some slight diffuse degenerative changes in the nuclei of the cerebral nerves, as well as in the nuclei of the vestibular nerve, but without any characteristic signs. Concluding, we must, therefore, say that generalized brain pressure, as a rule, produces only very slight changes in the inner ear and in the centers of the eighth nerve. When we neglect the hernias of the brain, which, of course, do not influence the function of the inner ear, we must, therefore, expect only very slight ear symptoms in chronic generalized brain pressure. To be sure about this, I have chosen six cases, which had all the signs of an intensive generalized brain pressure, but without any localized brain pressure, i.e., without any brain tumor.

We found in all six cases a marked choked disc (till 5D). In five of these six cases, the roentgenographic findings were also markedly characterized by extensive destructions about the base and in the roof of the skull. Therefore, all six cases had a marked generalized brain pressure, although no tumor was present.

So far as the ear findings in these six cases are concerned, it is surprising that spontaneous dizziness, which is such a common symptom in all possible brain tumors, was absent. Also, spontaneous nystagmus and spontaneous disturbances in equilibrium were either completely or nearly completely absent. But most interesting is the fact *that four of these six cases gave quite normal responses to the caloric test.* Only two cases showed hyperirritability of even slight degree. Finally, also, the hearing was normal in all six cases. We have, therefore, to say: *In cases with chronic generalized brain pressure, but without localized pressure of the brainstem, the hearing stays normal and our method of caloric test gives either normal or slightly increased response. Therefore, the spontaneous labyrinthine symptoms (dizziness, nystagmus, disturbances in equilibrium) are in these cases either completely or practically absent.*

We see that in cases with chronic generalized brain pressure the functional test of the ear after our methods gives the same poor results as does the microscopic examination. That is the principal difference between ear and eye. Although generalized brain pressure produces marked changes in the anatomy and function of the eye, which we name "choked disc," the ear and its centres do not react at all or they react very little. *A choked labyrinth analogous to the choked disc, therefore, does not exist.*

TABLE II

CASE	DIZZINESS	COCHLEA	FALLING	NYSTAGMUS	CALORIC TEST	BILATERAL CALORIZATION	CHOKED DISC	ROENTGENOGRAMS	AUTOPSY FINDINGS
(A)	None	(?) Dementia	Left backwards	None	*Normal both sides*	None	+ +	+	Meningitis serosa obsoleta in regione bulbi cerebri
(B)	None	*Normal*	To the right	Rotary, Horizontal (I) to both sides	Both sides + left > right	Rotary, Horizontal (II) right	+ +	+ +	Meningitis serosa obsoleta
(C)	None	*Normal*	To the right	Rotary, Horizontal (I) to both sides, right > left	*Normal both sides*	None	+ +	+	Pseudotumor cerebri
(D)	None	*Normal*	None	None	*Normal both sides*	None	+ +	+ +	Cysticercus
(E)	None	*Normal*	None	Rotary, Horizontal (I) to the right	Both sides + right > left	None	+ +	None	Cysticercus
(F)	None	*Normal*	None	None	*Normal both sides*	None	+ +	+	Hydrocephalus

B. Influence of Chronic Localized Brain Pressure on the Function of the Ear

Chronic localized brain pressure, as far as it influences the ear, will be produced by tumors in the posterior fossa. Of most practical importance are the acoustic tumors and the cerebellar tumors. It is only with these tumors that we will deal here.

To get a clear view, we first must describe again the *anatomic changes* (a) in the peripheral ear organ, and (b) in the centres of the eighth nerve produced by localized brain pressure.

(a) Localized brain pressure and the peripheral ear organ — I have studied microscopically sixteen temporal bones from eight cases. There have been four acoustic tumors, three cerebellar tumors, and one case with a cluster of cysticercus in the cerebellopontile angle combined with a carcinomatous meningitis. In all these cases we have seen changes, which we name after Manasse and Günther "*stagnation hydrops of the inner ear.*" This finding is to be explained in the following way: The mentioned tumors compress the two veins which accompany both aqueducts and which bring the blood out from the inner ear. In this way, transudates and hemorrhages in the spaces of the inner ear will be produced. These changes are essential for stagnation hydrops. This hydrops can disappear without sequellæ when it does not reach a high degree and when a decompressive operation early produces a diminution of stagnation. Also, the decreasing blood pressure before death allows the outflow of blood or transudate from the inner ear. Therefore, the hydrops in a specimen is, as a rule, less marked than is probably true during life. When the hydrops reaches a high degree and lasts a long time, which practically happens only in acoustic tumors, it heals like an inflammatory disease of the inner ear with development of connective tissue and bone which fills up a part of the spaces of the inner ear.

It is quite clear that this stagnation hydrops of the inner ear is not analogous to choked disc, but it is interesting that Graefe, who first described choked disc, explained his finding as a consequence of compression of the cavernous sinus, an opinion which today is at least not generally accepted.

A further consequence of chronic localized brain pressure is seen in changes in the lumen of the internal meatus, which are most intensive in acoustic tumors, but which we may find also

in cerebellar tumors. The internal meatus can become larger or narrower in these tumors. More often there is enlargement, which has been known for a long time. In such cases the walls of the internal meatus are destroyed by osteoclasts and by Pacchionian granulations.

Much less familiar, but practically very important, is the fact that in tumors of the posterior fossa the internal meatus can become narrower. This happens in the following way: When, for instance, an acoustic tumor lies in the cisterna pontis lateralis, which tumors we name "mesial acoustic tumors," it may irritate the periosteum of the internal meatus, which is in direct communication with the dura of the posterior fossa, so much so that this periosteum produces exostoses which make the canal narrower instead of larger.

The last consequence of localized brain pressure on the peripheral ear organ is *the sheath-hydrops of the eighth nerve*, which we have seen previously only in acoustic tumors. You know that the eighth nerve, before entering the internal meatus, lies free in the cisterna pontis lateralis, which is a cavity filled with cerebrospinal fluid in the arachnoidal space. As the nerve enters the internal meatus, the dura follows the nerve and covers the walls of the internal meatus as periosteum. The arachnoid also follows the nerve and covers it as a fine sheath. Now we will see later that an acoustic tumor produces, as a rule, a curve-like deformity of the brainstem, so that the cisterna pontis lateralis of the normal side lies just at the vertex of that curve. Therefore, the cisterna of the normal side will be compressed and a stagnation of cerebrospinal fluid in the cisterna of the normal side is the effect of that compression. This stagnation of fluid presses farther along the eighth nerve and removes the arachnoidal sheath from the nerve. We get, therefore, an expansion of the arachnoidal sheath on the normal side, which we name *sheath-hydrops of the eighth nerve*.

Microscopically we find a similar picture in the well-known sheath-hydrops of the optic nerve. We see that the thickened and slightly inflamed arachnoidal sheath is removed from the nerve and pressed against the periosteum of the internal meatus. It is quite clear that the sheath-hydrops of the eighth nerve never can reach such size as the sheath-hydrops of the optic nerve, because the orbit is much larger than the internal meatus. But the sheath-hydrops of the eighth nerve produces another

finding which is clinically important. We have seen that only a relatively small expansion of the sheath-hydrops of the eighth nerve is necessary to press the arachnoid against the periosteum of the internal meatus. The effect of that pressure is a destruction of the underlying bone. The sheath-hydrops may enlarge the internal meatus on the normal side, a finding that we see also, at times, in living patients with acoustic tumors by means of roentgenograms.

We see that chronic localized brain pressure works in quite a different way from generalized brain pressure. As a consequence of that localized pressure on the peripheral ear organ, we could find: (1) the stagnation hydrops, (2) the changes in the lumen of the internal meatus, and (3) the sheath-hydrops of the eighth nerve. It is, of course, quite clear that we do not find in every case of tumor in the posterior fossa all these changes. But when they are developed, they influence markedly the clinical examination of the ear.

(b) Localized brain pressure and centres of the eighth nerve — The brainstem, with its centres of the eighth nerve, can be compressed laterally by tumors of the cerebello-pontile angle, especially acoustic tumors, or it can be compressed from above in tumors of the fourth ventricle, especially cerebellar tumors. In the first circumstance, the brainstem may be pushed aside so that it makes a curve with the vertex in the cerebello-pontile angle on the healthy side. These conditions, following Cushing's monograph, are very well known. Since the blood vessels of the cerebello-pontile angle are very delicate, we see microscopically, in these cases, vascular abnormalities in the brainstem, such as œdema, softening, and hemorrhage.

In cerebellar tumors, which compress from above, the brainstem seems to be very resistant. We sometimes see in these cases the brainstem flattened, but when we examine it microscopically we find only slight diffuse, degenerative changes similar to generalized pressure, only more marked. That is, of course, surprising, because oftentimes in such a case the patient has died of respiratory paralysis. To explain this difference between clinical and microscopical findings, we must say that the usual methods of staining are not sufficient to demonstrate all changes in the brainstem, which are after the clinical findings surely present also in these cases.

Contrary to these cases, we sometimes see the most severe degeneration when the brainstem is very intensively compressed from above. We find in such cases severe destruction and necrosis in the ganglion cells; furthermore, an extensive destruction of myelin sheaths in the fibres of the brainstem. I have examined thoroughly two such cases.

The marked changes in the inner ear and in the central pathways of the eighth nerve produced by chronic localized brain pressure, of course, influence the clinical findings. When we first recall the changes in the peripheral ear organ, we find that chronic localized brain pressure can make the internal meatus larger and narrower, which happens mostly on the side of the tumor, sometimes on the normal side when a sheath-hydrops is present. All these possibilities we have to know when we want to explain a roentgenogram of such cases in the right way.

Of further importance are the clinical consequences of stagnation hydrops of the inner ear. It is quite clear that the fully developed stagnation hydrops must diminish the hearing and the caloric irritability of the ear. But since we have learned that stagnation hydrops may afterwards disappear without remains, it follows that in such cases the diminution of hearing and caloric irritability is reversible when the stagnation hydrops disappeared. In that way we can explain why the diminution of hearing in cerebellar tumors sometimes shows variations in intensity and why sometimes the hearing can improve after decompression. All these findings are, of course, impossible when the stagnation hydrops has produced a development of connective tissue or bone in the spaces of the inner ear. In these cases the impairment of function is persistent.

All these changes in the inner ear are very important to explain the otological findings in acoustic tumors. They are a little less important to explain the otological findings in cerebellar tumors. In these last mentioned cases, the anatomic changes, which are produced by chronic localized brain pressure in the *brainstem*, are of more importance than the changes in the inner ear. We have, therefore, to answer the question: How will the functional tests of the ear be influenced by localized pressure pressing against the brainstem from above, i.e., from the fourth ventricle? We shall try to answer this question on the basis of our own examinations, which are, of course, not definite as long as they are not proved by others.

To get a clear view of how the centres of the eighth nerve in the brainstem react against pressure, we have first to remember that the different centres in the brainstem, such as the heart centre, the vasomotor centre, or the respiratory centre, show a different sensibility to pressure. One reacts earlier than the other; but they also react in a different manner, either with irritative or with paralytic symptoms. That is dependent on the duration and intensity of the pressure. Since the centres of the eighth nerve lie in the brainstem, very near to the earlier mentioned centres of the vagus and sympathetic nerve, we have to ask, in which way do these centres of the eighth nerve react against pressure?

As far as generalized pressure is concerned, we have already mentioned that it does not evoke any symptoms from the cochlear centres. Also the vestibular centres do not react at all or only with slight irritative symptoms, especially with slight hyperirritability. Quite different conditions are found in localized pressure. First, we must say *that the vestibular centres, especially the centres for caloric nystagmus, are much more sensitive to localized brain pressure than the cochlear centres.* Therefore, in localized pressure, we get vestibular symptoms much earlier than cochlear symptoms. In cases with slight or moderate degrees of pressure, these vestibular symptoms are, of course, first irritative symptoms, which clinically clearly appear as labyrinthine hyperirritability. Really, in this series of thirty-two cases of infratentorial tumors that I have shown, 56 per cent had labyrinthine hyperirritability in the preoperative stage and only 31 per cent showed a normal response to the caloric test. In all these cases the hearing was normal. Only cases with metastases of malignant tumors in the posterior fossa or cases which had, along with the tumor, a severe nutritional disturbance of the brain, especially an arteriosclerosis, only these cases are exceptions, because in them we may find instead of a hyperirritability a hypoirritability of the labyrinth. But neglecting these cases, we can say that *the first clinical effect of localized pressure upon the brainstem in cerebellar tumors is a labyrinthine hyperirritability with normal hearing.* Microscopically, we find in these cases the slight degenerative changes in the brainstem earlier mentioned, which, of course, do not show any signs significant of localized pressure.

To prove our viewpoint we wish to emphasize these facts:

(1) As we have already mentioned, in cases with generalized brain pressure but without localized pressure, we find either normal irritability or a very slight degree of hyperirritability. The last finding is rare; therefore, generalized pressure does not produce hyperirritability of higher degree.

(2) As far as the infratentorial tumors with normal irritability are concerned, we find that cerebellar tumors which do not produce localized brain pressure of higher degree, such as small cysts on tubercles, likewise do not produce hyperirritability.

(3) Of importance is the influence of an operation on labyrinthine irritability. Here we can say that simple decompression without removal of the tumor does not influence the labyrinthine irritability at all or is only transitory in effect, because such an operation can only diminish generalized pressure, but not localized pressure. On the contrary, we sometimes see in rapidly growing cerebellar tumors that in spite of decompression the increasing localized pressure produces a paralysis of vestibular centres and, therefore, a hypofunction of labyrinths. But when it is possible radically to remove a benignant cerebellar tumor, we often see that the preoperative hyperirritability is slowly changed into a postoperative normal irritability. As a rule, when a radical removal of cerebellar tumor is not possible, the labyrinthine hyperirritability persists or may be changed into a hypofunction of labyrinth.

(4) Evidence for support of our opinion was found in two cases which showed very well the changes of the function of the ear in increasing localized brain pressure. You will, therefore, permit me to tell you a little more about these cases.

The first case was that of a girl, ten years of age, who came to the department with the diagnosis of Encephalitis lethargica. The autopsy disclosed a closure of the foramen Magendi with huge enlargement of all ventricles. The fourth ventricle, especially, showed a balloon-like enlargement, which produced an increasing localized pressure upon the brainstem. About one month before death, we found in this case a normal hearing and a very clear bilateral hyperirritability. Eighteen days later the hearing could not be examined perfectly, but surely no deafness was present. However, we found on that day, on both

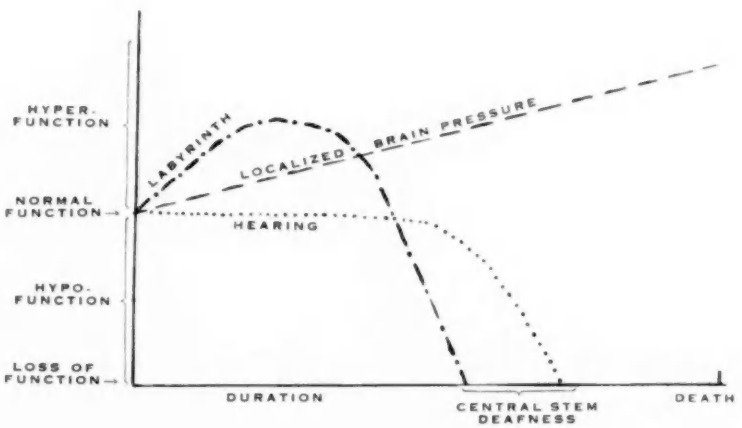
sides, a labyrinthine hypoirritability in the sense that we could evoke from both sides only the slow component of nystagmus. Twelve days later, i.e., 30 days after the first ear finding and one day before death, deafness was still not present, but it was questionable if the hearing was still normal. Anyway, on that day neither labyrinth gave any response. The microscopic examination in that case showed the most severe degree of degeneration in all nuclei of the brainstem, but the inner ears were normal.

The second case was that of a man with an enormous glioma infiltrating practically the entire cerebellum, which produced a very intensive localized pressure on the brainstem. The case was under observation one and a half years and showed at first normal hearing and normal irritability. Decompression was performed without removal of the tumor. About one year after the first ear examination, and about ten months after decompression, hearing and labyrinths were still normal. But three months later we found a high degree of labyrinthine hypoirritability on both sides. The hearing was still normal. The cerebellum was again exposed, but the tumor was not removed. Two months after that operation, and 10 days before death, we found deafness on both sides and the labyrinths were not irritable at all. Therefore, in that case during three months the normal hearing was changed to deafness and the labyrinthine hypoirritability was changed to a non-irritability. The microscopic examination showed that in the brainstem the greatest part of the fibres, of course, also, the pathways of the vestibular and cochlear nerves, were also demyelinized. The cochleas on both sides showed a slight descending degeneration.

These two cases are principally important because they show in an excellent manner the influence of increasing localized pressure upon the function of the ear. About this we can therefore say: When the localized pressure increases, the centres of the eighth nerve change from the irritative condition to the paralytic condition, in the same way as, for instance, the vasomotor centres do. We always find this change in the vestibular centres earlier than in the cochlear centres. Clinically, we see, therefore, that with increasing localized pressure, first the hyperirritability is changed to hypoirritability, and finally to non-irritability.

In the stage of hyperirritability the hearing is always normal, unless some peripheral lesion disturbs the hearing. Only when the hyperirritability changes to a hypoirritability does the hearing become disturbed. This disturbance of hearing can increase to complete deafness when the localized pressure increases and the labyrinthine function decreases further. The anatomic basis for that finding we know. You have seen just now that there are several degenerative changes in the brainstem.

It is quite clear that we must separate the deafness produced by increasing localized pressure from other kinds of central deafness, and we, therefore, call it "*central brainstem deafness*" or shortly "*central stem deafness.*" Clinically, this central stem deafness is characterized by the following findings: (a) It is combined with hypofunction of labyrinth, (b) it shows a rapid progress, (c) it appears very late, (d) it is combined with pontine and medullary symptoms, and (e) it will be produced by diseases which press in highest degree from above, i.e., from the fourth ventricle against the brainstem. When we finally ask how the function of both parts of the inner ear will be changed in increasing localized pressure, the following diagram may give the answer.



This diagram shows that the localized pressure produces for a long time a labyrinthine hyperirritability with normal hearing. In that stage we examine the largest number of patients who have cerebellar tumors. Much later the hyperfunction of

the labyrinth changes to hypofunction, and still later the normal hearing changes to deafness. Finally, shortly before death, we get the fully-developed finding of central stem deafness.

Until now we have always mentioned that we find most often, in infratentorial tumors, a labyrinthine hyperirritability with normal hearing. The question arises, is that hyperirritability unilateral or bilateral? This question we can not answer with certainty, because cerebellar tumors which produce a sharply localized pressure on one side only are rare. We, therefore, get the impression that the hyperirritability on both sides is most often equal when we use the *unilateral calorization.*

We get quite another finding when we use *bilateral calorization.* With this method we found that in twenty-eight cases of infratentorial tumors, fifteen cases (53 per cent) showed difference in the irritability of the two sides. This difference was demonstrated by nystagmus of II degree, which appeared after bilateral calorization. We therefore must say, *in the majority of infratentorial tumors we find a difference in the irritability of the two sides, also demonstrated by a positive result in bilateral calorization.* Therefore, the results of unilateral and bilateral calorization in infratentorial tumors are not always in concordance. In the relatively rare cases, in which a cerebellar tumor lies circumscribed on one side only, the nystagmus after bilateral calorization, as a rule, goes to the affected side, which means a higher degree of irritability on the normal side.

When we now ask the reasons for all these symptoms, we must say that when we perform bilateral calorization in a normal individual and in the normal position of the head, we do not evoke any nystagmus. In that case the equilibration centres of the vestibular nerve, on both sides, are irritated by the labyrinthine impulse at the same time and in the same degree, but in opposite directions. These oppositely-directed irritations neutralize another already in the region of vestibular nuclei, so that no impulse can reach the eye muscle nuclei. Bilateral calorization, therefore, does not give a response. Quite different are the conditions when the vestibular centres already are spontaneously irritated by localized pressure or for some other reason. It rarely happens, of course, that a morbid factor produces a bilaterally symmetrical effect. We, therefore, can only

occasionally expect that spontaneous irritation of the vestibular centres will reach exactly the same degree on both sides. Much more often the irritative condition will be more intensive on one side than on the other. When we perform bilateral calorization in such a case, we increase the difference in the spontaneous irritation of the vestibular centres on both sides. Therefore, no neutralization can occur between the oppositely-directed impulses in the vestibular nuclei on both sides. Therefore, an impulse will be produced in the vestibular centres which reaches the eye muscle nuclei and this impulse produces nystagmus. Bilateral calorization, therefore, gives response.

It follows that bilateral calorization responds easiest when labyrinthine hyperirritability is present. Clinical experience is in accordance with that. Indeed, bilateral calorization, as a rule, does not give response in all cerebellar tumors which show a hypoirritability on both sides with unilateral irrigation and in cerebellar tumors which grow by substitution and which do not produce localized pressure of higher degree.

Summary

Summarizing, we can say:

(1) Chronic generalized brain pressure does not produce marked changes either in the inner ear or in the central pathways of the eighth nerve.

(2) Therefore, functional tests of the ear will never be markedly changed by chronic generalized pressure.

(3) Chronic localized brain pressure may produce the following changes in the temporal bone: (a) a stagnation hydrops of the inner ear which can again disappear without remains or which can heal with the development of bone and connective tissue in the spaces of the inner ear; (b) changes in the size of the internal meatus on the diseased or on the healthy side. Both changes are frequent and marked in acoustic tumors. They are less frequent and less marked in cerebellar tumors.

(4) Chronic localized brain pressure of low degree and short duration produces slight, diffuse, degenerative changes in

the central pathways of the eighth nerve. Chronic localized brain pressure of high degree and long duration produces most severe degenerative changes in the ganglion cells and in the fibres of the central pathways of the eighth nerve.

(5) The clinical consequences of changes in the temporal bone are changes in the acuity of hearing, as we find it sometimes in acoustic tumors on the healthy side and in cerebellar tumors. Furthermore, the changes in the size of the internal meatus, which can be recognized by roentgenograms. All these changes in the temporal bone are analogous to the primary atrophy of optic nerve produced by localized pressure.

(6) As clinical consequences of the changes in the central pathways of the eighth nerve we find in localized pressure of low degree and short duration a labyrinthine hyperirritability, positive response of bilateral calorization, and normal acuity of hearing. By increasing localized pressure the labyrinthine hyperirritability will be changed to hypoirritability and finally to nonirritability, and at the same time, the normal hearing will be impaired until deafness results. This final consequence of localized pressure, coming from the fourth ventricle pressing against the brainstem, we name "central stem deafness."

(7) The difference in the reaction of ear and eye to increased brain pressure is the following: The eye reacts to generalized brain pressure as well as to localized pressure. In the first condition, a choked disc; in the second condition, primary atrophy of optic nerve may be produced. The ear does not react to generalized brain pressure at all; to localized brain pressure, it may react with symptoms which can be clinically recognized by the functional test as well as by roentgenograms.

(8) The secondary pathways of the optic and of the eighth nerves both respond to generalized pressure with very few symptoms or no symptoms at all, but both pathways react to localized pressure. The difference is only that the secondary pathways of the vestibular nerve, when they are pressed, produce very early a labyrinthine hyperirritability, although the optic and cochlear pathways react much later, with defects in the visual field and defects in hearing.

Gentlemen, I have spoken long, very long, too long. I beg your pardon. I have to do that because, unfortunately, the abundance of words is in opposite relationship to the abundance of definite knowledge. However, that can not be changed, since the contributions of otology to neurology are very rich insofar as theoretical deductions and experimental work are concerned, but they are very poor insofar as definite anatomical facts come into question. But, I think, just the anatomy shows the way we have to go in order that otoneurology may become general knowledge among all doctors as ophthalmoneurology already has been for many years. The American ear, nose, and throat specialists, with their enormous scientific power and energy, are able to get to this new land of medicine, which I have seen today only from afar, as Moses saw the holy country.

THE HISTORY OF THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

MAX A. GOLDSTEIN, M.D.*

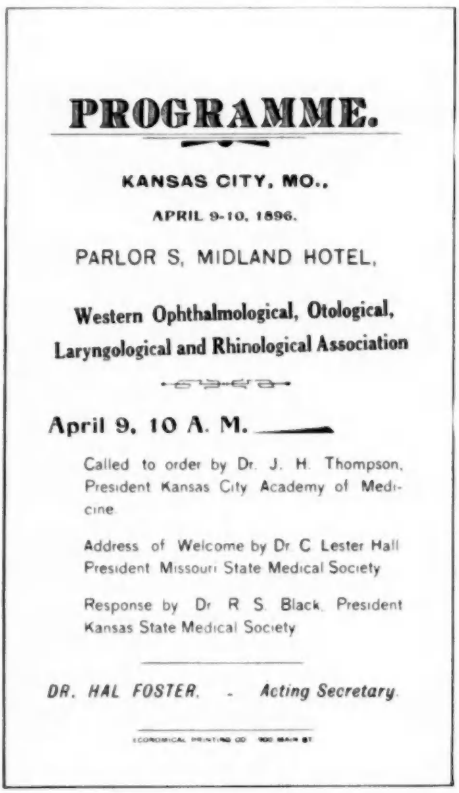
ST. LOUIS

TO CHRONICLE the history of the Academy may be an exalted task, but there is scant opportunity for flights of oratory or flow of wit, yet history and historians are necessary adjuncts to organized society and civilization; so I will ask your indulgence for the necessary data injected into this chronicle.

When President Eagleton invited me to present a sketch of the history of this organization he honored me as a charter member and as an early President of the Academy and stimulated my memory of its inception nearly forty years ago; he also honored my native state of Missouri, since this Society was born there in 1896.

While the Academy is celebrating its Silver Anniversary tonight, we must not fail to respect its ancestry and glorify the genealogical tree from which it sprang. To Dr. Hal Foster, of Kansas City, belongs the credit for its birth. In the fall of 1896, there was issued by Dr. Foster, as acting secretary, an invitation to a selected group of Eye, Ear, Nose and Throat Specialists of the West and the Southland to meet in Kansas City to organize such an association, and this meeting was held April 9 and 10, 1896, in Parlor S of the Midland Hotel. The meeting was called to order by Dr. J. H. Thompson, President of the Kansas City Academy of Medicine; the address of welcome was given by Dr. C. Lester Hall, President of the Missouri State Medical Association, and the response by Dr. R. S. Black, President of the Kansas State Medical Society. About fifty physicians were

* Address given at the Banquet Session, Wednesday evening, September 18, 1935. Dr. Goldstein is the oldest living past president of the Academy.



PROGRAMME.

KANSAS CITY, MO.,

APRIL 9-10, 1896.

PARLOR S, MIDLAND HOTEL,

Western Ophthalmological, Otological, Laryngological and Rhinological Association

April 9, 10 A. M.

Called to order by Dr. J. H. Thompson, President Kansas City Academy of Medicine.

Address of Welcome by Dr. C. Lester Hall President Missouri State Medical Society

Response by Dr. R. S. Black, President Kansas State Medical Society

DR. HAL FOSTER, - Acting Secretary.

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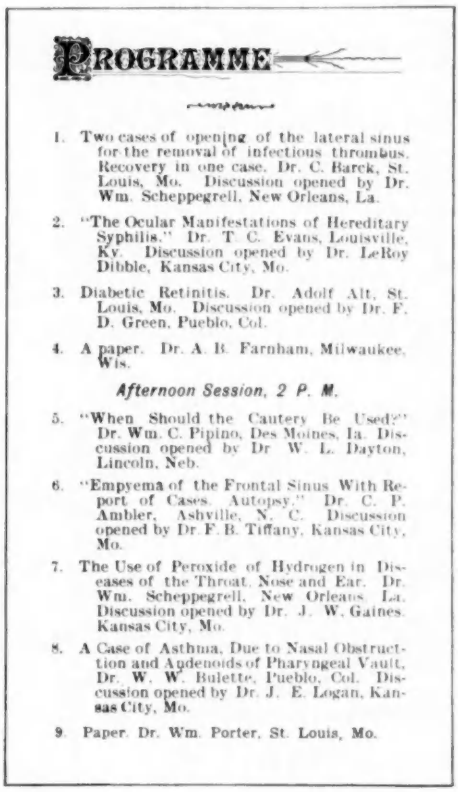

PROGRAMME

1. Two cases of opening of the lateral sinus for the removal of infectious thrombus. Recovery in one case. Dr. C. Barck, St. Louis, Mo. Discussion opened by Dr. Wm. Scheppegrell, New Orleans, La.
2. "The Ocular Manifestations of Hereditary Syphilis." Dr. T. C. Evans, Louisville, Ky. Discussion opened by Dr. LeRoy Dibble, Kansas City, Mo.
3. Diabetic Retinitis. Dr. Adolf Alt, St. Louis, Mo. Discussion opened by Dr. F. D. Green, Pueblo, Col.
4. A paper. Dr. A. B. Farnham, Milwaukee, Wis.

Afternoon Session, 2 P. M.

5. "When Should the Cautery Be Used?" Dr. Wm. C. Pipino, Des Moines, Ia. Discussion opened by Dr. W. L. Dayton, Lincoln, Neb.
6. "Empyema of the Frontal Sinus. With Report of Cases. Autopsy." Dr. C. P. Ambler, Ashville, N. C. Discussion opened by Dr. F. B. Tiffany, Kansas City, Mo.
7. The Use of Peroxide of Hydrogen in Diseases of the Throat, Nose and Ear. Dr. Wm. Scheppegrell, New Orleans, La. Discussion opened by Dr. J. W. Gaines, Kansas City, Mo.
8. A Case of Asthma, Due to Nasal Obstruction and Adenoids of Pharyngeal Vault, Dr. W. W. Bulette, Pueblo, Col. Discussion opened by Dr. J. E. Logan, Kansas City, Mo.
9. Paper. Dr. Wm. Porter, St. Louis, Mo.

present, and it is interesting to note that the following twenty-seven states were represented: Alabama, Arizona, Arkansas, California, Colorado, Florida, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Michigan, Minnesota, Mississippi, Missouri, Montana, Nebraska, North Carolina, Ohio, Oregon, South Carolina, Tennessee, Texas, Virginia, West Virginia, and Wisconsin.

Dr. Adolph Alt, of St. Louis, was elected the first President, and Dr. Hal Foster, Secretary. The Society was named the Western Ophthalmological, Otological, Laryngological and Rhinological Association. But little time was spent in discussing Constitution, By-Laws, and red-tape, so usual at assemblies of this character. An elaborate prearranged scientific program occupied the time of a full two-days' session. A printed copy of the original program is here offered as a souvenir of the occasion.

10. Tinnitus Aurium. Dr. J. W. May, Kansas City, Kan. Discussion opened by Dr. Adolf Alt, St. Louis, Mo.

11. Paper. Dr. Robert Leavy, Denver, Col.

Evening Session

12. Panopthalmitis Caused by Leucoma Adherens (Anterior Synechia) and the Treatment of Anterior Synechia. Dr. L. A. Lebeau, St. Louis, Mo. Discussion opened by Dr. J. E. Minney, Topeka, Kan.

13. Tubercular Laryngitis at High Altitudes. Dr. B. P. Anderson, Colorado Springs, Col. Discussion opened by Dr. D. Milton Green, Grand Rapids, Mich.

14. Why Deafness Afflicts Some and Not All Who Suffer from Diseases of the Upper Air Passages. Dr. W. F. Strangways, Flint, Mich. Discussion opened by Dr. G. A. Wall, Topeka, Kan.

15. Purulent Brain Infection from Otitis Media. Dr. B. E. Fryer, Kansas City, Mo. Discussion opened by Dr. C. Barck, St. Louis, Mo.

Morning Session.

16. Subjective Sensations. Dr. W. E. McVey, Topeka, Kan. Discussion opened by Dr. John C. Smith, St. Louis, Mo.

17. Which Operation for Senile Cataract Should the Beginner Select? Dr. J. H. Thompson, Kansas City. Discussion opened by Dr. T. C. Evans, Louisville, Ky.

18. The Clinical Examination of Deaf Mutes. S. T. Walker, Jacksonville, Ill., Superintendent of Illinois State Institution of the Deaf and Dumb. Discussion opened by H. C. Hammond, Superintendent of Kansas State Institute for Deaf and Dumb, Olathe, Kan.

19. Usual Head Formation With Eye, Nose and Throat Complications. Dr. H. W. Woodruff, Joliet, Ill. Discussion opened by Dr. R. S. McGee, Topeka, Kan.

20. Adenoid Vegetations of the Vault of the Pharynx—Report of Cases. Dr. C. E. Clark, Kansas City. Discussion opened by Dr. M. F. Jarrett, Fort Scott, Kan.

21. A paper. Dr. John A. James-James, St. Louis, Mo.

22. Errors in the Literature on Javal's Ophthalmometer for the Measure of Astigmatism. Dr. G. W. Grove, Kansas City, Mo. Discussion opened by Dr. W. A. Shoemaker, St. Louis, Mo.

Afternoon Session, 2 P. M.

23. The Present Status of the Physiological Anatomy of Vocalization and Phonation. Dr. Edward H. Schaefer, Kansas City, Mo. Discussion opened by Dr. H. W. Loeb, St. Louis.

24. Eye Affections as Related to Nasal Diseases. Dr. George E. Bellows, Kansas City, Mo. Discussion opened by Dr. E. E. Hamilton, Wichita, Kan.

25. Organization a Protecting Factor for the Up-to-date Physician. Dr. J. D. C. Hoit, Elmwood, Ill. Discussion opened by Dr. B. P. Anderson, Colorado Springs, Col.

26. A paper. Dr. H. W. Loeb, St. Louis.

27. Goitre. Dr. G. J. Cullen, Cincinnati, O. Discussion opened by Dr. W. F. Strangways, Flint, Mich.

28. The Relation of Opticians to Ophthalmology. Dr. F. D. Green, Pueblo, Col. Discussion opened by Dr. H. L. Gill, Pittsburg, Kan.

29. Eye Symptoms of Brain Tumor. Dr. J. M. Ball, St. Louis, Mo. Discussion opened by Dr. G. W. Gore, Kansas City, Mo.

The members present signed the roster and this was recognized as the charter membership of the organization.

Time has rolled on and taken its toll, and of this original group of fifty, only six members survive: Doctors Carl Barck, St. Louis; Hamilton Stillson, Seattle; Herbert Moulton, Fort Smith, Arkansas; Fayette C. Ewing, Alexandria, Louisiana (then of St. Louis); Hal Foster, Kansas City, and Max A. Goldstein, St. Louis.

The complexion of the membership then was distinctly western and southern in character and it maintained this geographic limitation for a number of years, until its rapid growth and scientific activities attracted ophthalmologists and otolaryngologists of all sections of the country.

The senior national societies in these specialties, the American Ophthalmological, the American Otological, the American Laryngological, and the American Laryngological, Rhinological and Otological Societies, had prescribed limitations in their membership to which younger men of the profession were rarely eligible.

The outstanding purpose of this Society then was to offer the younger men the opportunities of a scientific forum and fellowship, and this has continued as a valuable tradition of this organization.

Coincident with the celebration of the Silver Anniversary of the Academy today and the meeting held in Cincinnati in 1901, during my presidency, the idea of changing the name of the organization was first discussed, and in 1910, at the meeting in Cincinnati, the name was changed from the Western Ophthalmological, Otological, Laryngological and Rhinological Association to the American Academy of Ophthalmology and Otolaryngology. The membership had increased to over seven hundred, the geographical distribution of the membership had extended to every state in the Union and these united forces clamored for and were justified in their plea for national recognition and a national name; and so this sturdy and lusty son of a pioneer Missouri sire was born.

As the scientific strength of the Academy grew and its membership increased by leaps and bounds, another valuable feature was added as the demand by the younger element for postgraduate study became insistent. The promoter of this feature was Dr. Secord Large of Cleveland. In 1921, at the meeting in Philadelphia, the first Section on Postgraduate Instruction was announced as a part of the annual program. These Study Groups, led by selected members who had specialized in the specialties, became so popular and were so well attended that they have been one of the outstanding activities of the organization and have been continued with ever-increasing interest and efficiency to the present day. An annual meeting of the Academy today without these formidable Study Groups of Instruction Courses would lack one of its most valuable units, — and well might this be so, for in the growth, evolution and independence of American Ophthalmology and Otolaryngology and the conflicts of European strife and entanglements, the foreign meccas for further study

in our specialties are no longer indispensable and the exodus of American specialists to Europe less frequent.

Our own universities, in their medical departments, have rightfully assumed their responsibilities and interests in the teaching of the specialties, and America today has its own meccas and centers of Ophthalmology and Otolaryngology.

With the organization and practical operation of the American Board of Ophthalmology and the American Board of Otolaryngology, the Academy has made its most valuable contribution not only to American Ophthalmology and Otolarynogology, but also to American medicine.

This thought originated in the American Ophthalmological Society and was actively followed up by the Academy. To Doctors Beck, Carmody, and Skillern must be given credit for its impetus in the Academy — all men of fine executive talent, foresight, scientific optimism, and constructive purpose.

It was a glorious piece of work from its inception to the practical development of its present efficient form. What it has done and will do to elevate the standards and maintain the scientific strength and professional dignity of American Ophthalmology and Otolaryngology is an unwritten chapter in medical history.

To those who are more intimately informed concerning the work and purpose of the groups constituting these examining boards, their conscientious, loyal, impartial, and efficient service, the widespread value of scientific standards created by these labors, the legitimate safeguarding of medical practice and of the best interests of patients, stand out as magnificent factors of human and humane accomplishment.

Step by step the work of these examining boards marches on to greater heights; foundations stones are being laid for the building of a finer and better American Ophthalmology and Otolaryngology; other national organizations in the specialties have joined in the medical onward march; other specialties, realizing the value and character of these efforts, are adding their strength; hospitals, recognizing the influence and advantages of this protection to them and their patients, are limiting their staffs to qualified holders of certificates of these boards, and the outlook for a greater American medicine is becoming more hopeful.

Distinguished foreign specialists have been guests of the Academy every year and have contributed of their wisdom and experience to the scientific strength of our meetings.

The establishment of a research fund several years ago will assist those of our members whose talents incline toward original investigation to carry on. The printed Transactions of the scientific proceedings of the Academy each year have long since been recognized as a valuable contribution to the literature of our specialties.

At this interval I desire to offer a memorial tribute to three former Presidents of the Academy, all of Cincinnati, who have contributed much to the development of our specialties not only in their home city but to the nation at large. They are: Dr. Christian R. Holmes (President, 1902); Dr. Derrick Vail (President, 1908), and Dr. John W. Murphy (President, 1913).

It is not customary in a chronicle of this character to single out the services of any one member, but we should not overlook this opportunity to refer to the invaluable services that have been rendered for over ten years by our executive secretary, Dr. William P. Wherry. He has thoroughly justified the act creating the office of executive secretary, for he is a tower of strength in the discharge of his multiple duties in this office. It is due largely to his genius, alertness, and executive ability that the Academy has prospered in its numerical and financial strength and enthusiastic cooperation to so high a degree.

In forecasting future activities of the Academy we must not fail to cite: (1) Its attitude to State Medicine and the conservation of the equity and dignity of medical practice, (2) its influence in hospital control of qualified special practice, (3) regulation of clinic abuses, (4) cooperation with the Permanent Committee on the Deaf Child appointed by the American Medical Association about ten years ago, (5) helpful service to Leagues for the Hard-of-Hearing throughout the country, (6) prosecution and elimination of quacks and quackeries, and (7) participation in state and national legislation in the interests of American Medicine.

If we may judge the future of the Academy by its accomplishments in the past, it may be fair to predict that the continuance of its activities, its moral influence for better medical practice, its scientific democracy, and its united strength will yield a golden harvest in the interests of mankind.

SYMPOSIUM ON HEADACHE

* * *

A. FROM THE STANDPOINT OF THE OTOLARYNGOLOGIST

HARRIS H. VAIL, M.D.
CINCINNATI

PERHAPS the discussion of headache from the standpoint of the otolaryngologist can be introduced by presenting a simple classification. Such a one could be (1) Primary, Secondary and Reflex Neuralgia. (2) Ophthalmic Migraine. (3) Primary, Secondary and Reflex Headache.

Primary neuralgias and headaches are due to pathology within the cranial cavity and are the result of lesions directly affecting the sensory nerves or causing changes in the blood supply of the brain and drainage of the cerebrospinal fluid.

Secondary neuralgias and headaches are due to pathology adjacent to the brain or central nervous system. In this group the irritation, inflammation or suppuration, in the beginning at least, is localized to structures outside of the cranial cavity. Tumors, inflammation and suppuration of the nasal accessory sinuses, the orbit and its contents, the middle ear and mastoid cells and all other portions of the head are causative factors.

Ophthalmic migraine is not considered in this discussion.

Reflex neuralgias and headaches comprise that group wherein the etiological factor is at variable distance from the site of the headache even though there may be secondary alterations in the cerebral circulation and pressure. In this group are placed the headaches and neuralgias of allergic and toxic origin, of infectious and febrile diseases, of uremia, of poisons, or sepsis, and of disturbances of internal secretions.

The otolaryngologist is chiefly concerned with secondary neuralgias and headaches and my discussion deals with these groups, with the exception of those due to ocular conditions.

I am attempting to bring you, in what I hope is a practical way, the opinions of one who, while in medical school, was a student interne in a psychopathic institute, after graduation an interne at the Peter Bent Brigham Hospital where daily all kinds of brain tumors were encountered, then an interne at the Massachusetts Eye and Ear Infirmary, later an associate with his father, an ophthalmologist, where many patients complaining of ocular headache were seen, and finally at the present time an otolaryngologist in private practice. I can remember when almost every patient with headache I saw was a presumptive case of dementia præcox or some form of mental disease. Later on, a brain tumor was suspected. Now I have to be careful that I do not consider all headaches due to sinus trouble. I can assure you that it is very easy to fall into the error of regarding every case of headache as being in one's own particular specialty. Often that fallacy is initiated by the patients themselves. Perhaps the ophthalmologist is the easiest victim because his patients come to him of their own accord with a self-made diagnosis of an ocular headache. Due to education or perhaps to the standing of otolaryngologists more people are coming to them with a self-made diagnosis of sinus trouble. If it is important not to routinely consider every case of headache as due to sinus or tonsil disease, it is just as important not to be content with a routine examination of your patient. Be well assured that there are many conditions which are not easy to diagnose.

On the whole, problems of diagnosis of neuralgias and headaches from suppurative ear disease are not difficult, and it seems hardly necessary to do any more than mention the significance of deep-seated pain in the temple and orbital regions of the head. Such pain should never be disregarded at any time in a patient with present or very recent ear suppuration.

Inflammation in the superficial nasal or aural cavities causes superficial pain in the head, while inflammation in the posterior sinuses and deeper parts of the ear causes deeper pain. Contrast the headache and the exquisite tenderness of an acute suppurative maxillary-ethmoid-frontal sinusitis with the deep pain of an otitic subdural abscess or one of the petrous apex. Where there is involvement, irritation or inflammation of sensory nerves about whose origin and pathways and distribution there is not any dispute, the headache is definitely localized. Where the pathology involves nerves whose real functions may be somewhat unknown

at the present time (by that is meant nerves which combine a sensory and parasympathetic function such as the great superficial petrosal nerve), though the headache and neuralgia may be just as sharp or severe as in the former case, localization of the pain is not so definite. Very probably this is due to the fact that there are two distinct sensory systems in the face and head, one being the pathways of the 5th and 9th cranial nerves, the other being the system in which the great superficial petrosal is the chief element and which supplies deep sensation.

This leads me to the question, "What is the rôle of the cranial autonomic or parasympathetic or sympathetic system in the production of pain?" Clinical and experimental evidence have led to the belief that some rôle in the production of pain can be assigned to the sympathetic nervous system. It will require a great deal more work to be able to say with certainty just how this is done, but for practical purposes the sympathetic nervous system must be considered in any discussion on the subject of pain. This seems to have opened up a new field of diagnosis, prevention and treatment of diseases of the sympathetic nervous system as related to the ear, nose and throat, and I am sure that we as otolaryngologists will welcome attention and consideration of this problem.

Thus far I have not differentiated between headaches and neuralgia about the head. There is a difference between them. It is hard to describe, but according to my interpretation of the patients' description, neuralgia is much worse than headache and is associated with a depressed, worried sort of feeling, which the sufferer from headache does not have. It seems to me that neuralgia is about as severe as the pain in hemicrania or ophthalmic migraine. However, the latter is associated with nausea, vomiting and scotomata, which are not present in cases of secondary neuralgia of the head.

Frazier classified the neuralgias of the head as

1. Post-herpetic neuralgia.
2. Geniculate ganglion neuralgia.
3. Sphenopalatine ganglion neuralgia.
4. Glossopharyngeal neuralgia.
5. True major trigeminal neuralgia — Tic douloureux.
6. A large miscellaneous group classed as atypical neuralgia.

Cushing's classification of the neuralgias was

1. Neuralgias accredited to the sphenopalatine ganglion (Sluder's neuralgia).
2. Post-Zoster neuralgia.
3. Neuralgias accredited to the geniculate ganglion.
4. Painful tic.
5. Neuralgias from tumor involvement.
6. The minor trigeminal neuralgias. In this group were included cases where the nerves have been traumatized or have been involved by inflammation in the sinuses.

My classification is

1. Trigeminal neuralgia — Tic douloureux.
2. Vidian neuralgia.
3. Sphenopalatine ganglion neuralgia.
4. Tympanic plexus neuralgia.
5. Geniculate ganglion neuralgia.
6. Great superficial petrosal neuralgia.
7. Glossopharyngeal neuralgia.

Trigeminal neuralgia is not considered in this discussion.

Vidian neuralgia will be discussed later on.

Sphenopalatine ganglion neuralgia is rather rare and in my opinion is found only in cases where there is a tumor in the sphenomaxillary fossa or abscess in that locality, scars or pressure on the ganglion from hemorrhage due to instrumentation with a needle for local anesthesia or nerve block, and very rarely from fracture involving the superior maxilla. Strictly speaking, it may be improper to use the term sphenopalatine ganglion neuralgia as this ganglion is composed of sympathetic nerve cells and the exact rôle of the sympathetic nervous system in the production of pain is not known. For practical purposes, however, the term expresses very clearly the neuralgia produced by involvement of the various branches of the ganglion which are composed of a combination of fibers from the second division of the trigeminal, the great superficial nerve, and, of course, the fibers of the sympathetic nervous system in its general sense.

Geniculate ganglion neuralgia (including post-herpetic neuralgia) is not common. Through the kindness of Dr. H. P.

Mosher, I have been furnished with figures as to the incidence of herpes zoster of the ear at the Massachusetts Eye and Ear Infirmary from the years 1907 to 1933, inclusive. Of a total of 238,047 new ear cases, there were 23 cases of herpes zoster of the ear. The same relative scarcity of herpes zoster of the ear was reported from the Eye and Ear Infirmaries in New York. I have not been able to obtain any statistics as to the incidence of herpes zoster elsewhere about the head, but post-herpetic neuralgia or geniculate ganglion neuralgia can be regarded as very rare.

Tympanic plexus neuralgia certainly is rare.

"Great superficial petrosal neuralgia" is a creation of my own and may not be at all acceptable. This term was meant originally to designate only those cases of deep pain in the orbit, temple and about the ear in suppuration of the petrous apex. Now I should like to include the neuralgic pains occurring after section of the sensory root or removal of the gasserian ganglion for trigeminal neuralgia. Cases of apparent failure after a successful operation do occur. Dr. Alfred W. Adson in a personal communication stated that he would estimate roughly that about five per cent of trigeminal neuralgia cases had burning pains in the face and eyeball after a division of the sensory root. Dr. Harvey Cushing in a personal communication states, "In the old days there was a great deal of unnecessary tugging and pulling of the membranes which might easily have injured the superficial petrosal nerve and whether this is something which has been avoided in later years with more perfected approach, I can't tell." The usual procedure of elevating the dura over the front surface of the petrous bone also elevates and may tear the great superficial nerve resulting in scars and adhesions that can produce deep neuralgia in spite of the fact that there may be complete anesthesia over the distribution of the trigeminal nerve. That such damage does occur is suggested by facial paralysis following the operation where, very probably, the trauma to the great superficial petrosal nerve results in a hemorrhage in the geniculate ganglion. At this point, let me briefly review a case report. A woman, aged 66, four years previously began to have severe neuralgia in the right cheek, about the eye and forehead. After several years of this severe pain a chronic suppurative disease in the right maxillary sinus was diagnosed and a radical antrum operation was done with removal of diseased membrane, but without any relief of the neuralgia. A year later this was

diagnosed as tic douloureux involving all three divisions and the posterior root was divided by a competent neuro-surgeon. For two months there was complete relief of all pain. Then began a deep pain located in the right forehead, temple, back of the ear and particularly in and back of the eye, and the back of the head. This pain was described as being like needles and pins sticking the eye and pushing it out. In the past three months the pain has become quite severe and the patient is forced to resort to opiates for even a slight relief.

My examination showed the right cornea and cheek to be entirely anesthetic. Touching the right forehead and eyelid produced a spasm of pain, but pins could be stuck into this region without her feeling them. The nasal mucosa was anesthetic to the level of the mid-portion of the right middle turbinate. There was apparently no diminution of sensation over the front wall of the sphenoid and adjacent septum. There was an atrophic rhinitis of years standing on both sides of the nose. A few minims of 10 per cent solution of cocaine injected into the right sphenoid sinus through puncture relieved all pain except that in the upper eyelid and eyeball. Iodochlorol was injected into the right sphenoid and a roentgenogram made which showed an enormous sphenoid sinus with filling of a very deep lateral pterygoid recess and revealed an exceedingly close relationship between the sphenoid sinus and the vidian canal. The right antrum was quite cloudy on the roentgenogram. Thick pus was evacuated from the right sphenoid and maxillary sinus by irrigation. After six weeks of such treatment there was slight improvement in the pain in the back of the head and in the cheek but no relief from the pain in the eyelid and eyeball. Because the upper eyelid seemed to constitute a trigger zone, it was believed that some of the fibers in the ophthalmic division of the trigeminal nerve had regenerated. Consequently, the right supraorbital nerve was evulsed by the neuro-surgeon on June 15, 1935. This was followed by a definite diminution of pain in the eyelid and eyeball. However, pain in the nose and over the right cheek and once in a while in the back of the head continued, though not as severe as formerly. During this period the sphenoid and maxillary sinuses were being treated weekly by irrigation and instillation of various antiseptics. By the middle of August, five months after the first treatment, the sphenoid and maxillary sinus washings were clear. At this time the pa-

tient had gained a few pounds and, though not entirely relieved of pain, certainly was more comfortable.

This case could be discussed from a great many angles. I regard it as the toughest case I have ever seen. The following factors enter into the consideration of this case. First, a chronic suppurative maxillary and sphenoid sinus infection of years standing. Secondly, true trigeminal neuralgia. Thirdly, presumptive regeneration of some of the fibers of the ophthalmic division; and lastly, probable damage to the great superficial petrosal nerve from the operation upon the posterior root of the ganglion with subsequent neuralgia in this nerve. Whether the last factor actually exists is, of course, hard to say. The improvement in the deep pain following intrasphenoid therapy would seem to suggest the diagnosis of vidian neuralgia secondary to chronic suppurative sphenoiditis.

I feel that the most common form of neuralgia of the head is vidian neuralgia from sphenoiditis. Let me mention a few striking cases. I was asked by Dr. Samuel Seltz to see a patient who had been quite healthy up to a week before when following a cold "in the head" a headache began to bother him. Each day the headache had increased in severity, unrelieved by any sedative, including morphine. The headache was constant, severe, sharp and was not localized in any one place in the head. His temperature had been slightly elevated. His right maxillary sinus had been irrigated several times, pus appearing at each washing, but without affording any relief. The patient had become extremely restless and had to be kept under more or less restraint. When I entered his room at the hospital, he was sitting up in bed holding his head in his hands, moaning and tossing about. He was quite rational. The pain was worse on the right side and seemed to follow the typical distribution of vidian neuralgia. After cocainizing the nose a canula was passed into the sphenoid sinus by puncture of its anterior wall and at the moment of entrance there was an audible escape of air under pressure. The hiss was so loud that everyone in the room heard it. A few minims of 10 per cent solution of cocaine was injected and the sphenoid sinus irrigated. Within five minutes the patient experienced complete relief from pain. Later inquiry showed that he had soon fallen asleep. For the past year he has been entirely free

from headache. This was my first and only experience with the audible escape of air or gas under pressure from the sphenoid sinus.

Another case report is that of a 70-year-old country practitioner. He began four or five years ago to have constant, severe, agonizing headache on the right side. During the period he consulted many doctors. Anemia and evidences of a physical breakdown were found. His general condition improved under hospital treatment, but there was no improvement in the headache. He told me that he felt he could not go on any longer. Nasal examination did not disclose very much wrong. There was a little mucus to be seen in the back of the nose on the right side and the nasal mucosa was somewhat atrophic. However, as part of the examination, his right sphenoid sinus was punctured and radio-opaque oil was injected for roentgenographic visualization. He returned in a week saying that for the first time in years he was better. After five or six sphenoidal treatments he became much better. For about a year and a half he had no headache, but following a cold it returned. The same relief was experienced after a repetition of the sphenoidal therapy.

Another case, that of a doctor's daughter, presented some difficulties. My first impression was that she was having some sort of an endocrine disturbance. She said that hardly a day passed without there being a dull pain about the left side of the head. Sometimes there was a tight sort of feeling in the left cheek, left side of the nose and sometimes what she called headache. Whenever I can get a differentiation in the patient's mind between pain and headache, I am particularly pleased. Her last cold was four months previously, but questioning brought out that she noticed some post-nasal discharge and a mild stuffiness in the nose. She told me she had sinus trouble for years and had had an intranasal antrum operation four years ago with "hundreds of antrum washes," the last one being three weeks previously. Examination showed a high septal deviation to the right with pneumatic hypertrophy of the left middle turbinate. Both antra were bright upon transillumination. There was a good-sized opening into the left antrum. There was atrophy of the pharyngeal wall. The tonsils had been removed. Hearing was slightly diminished in the left ear. Iodochlorol was injected into the left

sphenoid sinus and roentgenograms were made which showed a very large sphenoid sinus projecting beyond the midline to the right with a very close relationship between the sinus and the left vidian canal. Roentgenogram of the left antrum showed no filling defect. Very slight improvement followed treatment of the left sphenoid sinus. Ergotamine tartrate was used with no improvement. After about a month, her father asked me to do the sphenoid operation. One year after operation she reported a complete relief from neuralgia. It might be argued in this case that the straightening of the septum and the removal of the large hypertrophied middle turbinate relieved a vacuum in the sinus which was the cause of her headaches. Maybe so, but unless a case diagnosed as vacuum headaches has received a thorough investigation of all the sinuses there will always be in my mind some doubt as to that diagnosis. The term vacuum headache is too broad in its scope and is too indefinite and misleading to be used with any accuracy.

The last case report is that of a thin, frail housewife who looked much older than the forty-six years she admitted. I first saw her in the summer of 1931. For four years she had been having, with increasing severity, attacks of pain coming on about once a week with a favorite time of onset around 3 a.m. The pain was on both sides of the head, but always worse on the left side. With every coryza she had pain. Besides, touching her face would bring on the pain. She said that the roots of her hair were very sensitive and that there was a feeling of needles and pins over the face. The pain was deep in back of the eyeballs and passed around to the left temple and ear. The attacks would usually last a day or two. She stated that she did not have headache. Examination showed that the nose was clear. There was a septal deviation to the right with extensive pneumatic hypertrophy of the left middle turbinate. My diagnosis was vidian neuralgia with trifacial neuralgia elements. Opaque oil was injected into both antra and roentgenograms made which showed no filling defects.

As cocainizing the front wall of her sphenoid always gave relief, I soon operated, resecting the nasal septum, trimming the left middle turbinate and removing a mucocele as large as a cherry which had dilated the bulla of the ethmoid. As I felt

that most of the pathology was coming from pressure exerted by the mucocele in the bulla of the ethmoid, I did not open the left sphenoid sinus. There was complete relief of pain for four months only. Since then, in the past four years, the attacks have come 2 to 5 times a year. Cocainizing the back part of the nose has given her relief each time. Adhesions in front of the sphenoid have prevented the probing of that cavity.

Inasmuch as post-operative adhesions always complicate the treatment of headaches from sinus disease, that question might be discussed now. Adhesions result from abrasion of two opposing mucous membranes in fairly close contact and usually follow the forcible use of the long Killian forceps and tight packing for post-operative hemorrhage. The best way to minimize the abrasion to the mucosa is to resect the septum as extensively as possible. This gives a better exposure and an easier operation upon the middle turbinate, sphenoid and the posterior ethmoid cells. Post-operative hemorrhage must be expected in most all cases. Even though no injury is done to the naso-palatine artery, when the packing is removed, a rather severe hemorrhage may follow and tight packing has to be resorted to. This last patient had such a hemorrhage from around the middle turbinate region and the nose had to be packed tightly for several days. Hardly ever is it necessary in doing the sphenoid operation in the ordinary case of vidian neuralgia to resect the floor of the sinus. I would compare surgery in that region to the refinements in operations on the maxillary sinus. In a radical antrum operation, it is not necessary to make such a large opening in the front wall of the antrum as to destroy many nerve fibers to vital teeth or to open up large arteries, and, besides, a radical antrum operation is not indicated in all cases of maxillary sinus trouble. The same principles hold in the treatment of sphenoid sinus infection.

These and other clinical experiences convince me that the sphenoid sinus should not be overlooked in the study of any case of headache. I do not want to bore you with a recital of vidian neuralgia from sphenoid disease. But I do want to say that experience with several hundred cases makes me feel confident that not only does this condition exist, but that it is fairly common, and that it can be easily relieved in a large percentage of cases. There have been no pathological studies made on the vidian nerve, only a few instances of study made on the spheno-

palatine ganglion, and then usually only in atrophic rhinitis, and only a few made of the geniculate ganglion. Once pathologists are stimulated to make routine studies of this part of the nervous system and their findings are correlated with the clinical findings of well-studied cases, real progress may be made in the problem of headaches.

Headache from infections of the other sinuses, I think, can be summed up in a few words. The same principles of diagnosis and treatment are applicable to all the sinuses. I believe that uncomplicated ethmoid infection is rare and it is hard to differentiate between headache which might come from an ethmoid infection alone and that which is due to a combination of maxillary sinus, ethmoid and resulting frontal sinus blockage.

Ophthalmic migraine is a subject in itself. It is a borderline condition between headache and neuralgia. There are many causes assigned to it. I think that most of its causes lie outside of our specialty.

I believe that the rôle of the otolaryngologist in the diagnosis of any case of headache is to study all the parts in his specialty with a thorough and impartial examination, placing the right amount of emphasis on the history and subjective findings. I think that he should always know the results of the examination made on the rest of the body before he gives his final opinion. It is perfectly possible to state at the first examination whether the patient has some pathology in the field of the specialty, but the important thing is to correlate the findings with those elsewhere in the body. A patient with headaches from lues of the central nervous system may have a cloudy antrum with pus in it. A patient with a brain tumor may have diseased-appearing tonsils. Hundreds of similar examples can be given. Often therapeutic tests must be resorted to. In other words, in a given case of headache, try the same treatment successfully used in positive cases of sinusitis. Within a week the results of the therapeutic tests can be checked. If the headaches are no better after nasal therapy, the cause for the headaches probably does not lie in the nose. In any case toxic enough to have headaches from tonsil disease, there would be evidences of toxemia elsewhere in the body, such as neuritis, arthritis or malaise. Headaches from disease of the middle ear and mastoid are found only when suppuration is present.

In closing, I would say that any one classification of headache is not satisfactory because its mechanism varies in a great many conditions and is at the present time not well known.

Inasmuch as there are so many different causes for headache, it is impossible for any one specialist to do more than to state the findings in his particular field of investigation. The final diagnosis depends upon the correlation of all the results of various examinations with proper consideration given to the various possible causes in the proper proportion of their importance and severity. Frequently, a therapeutic test of the nasal sinuses is required before a positive opinion should be given as to the importance of the sinuses in the causation of headache.

SYMPOSIUM ON HEADACHE

B. FROM THE STANDPOINT OF THE OPHTHALMOLOGIST

WILLIAM H. CRISP, M.D.
DENVER

IN WHAT MAY appear to some as the golden age of medical science, is it perhaps somewhat strange that the subject chosen for a symposium should be a mere symptom arising from a great variety of causes, yet whose precise nature and origin we do not understand? Who can accurately define pain? And who can accurately define headache, except by saying that it is pain in the head?

Pain may be thought of as Nature's warning that the sufferer is in danger from injury or disease. It is a sensory impression of disturbed vitality. It provokes immediate withdrawal of the damaged or threatened bodily part from the source of danger, or in disease it often serves to compel cessation of physical activity, in other words, to induce rest, which Hilton classically portrayed as the enemy of pain and disease. In functional gastrointestinal disorders it often contributes to the patient's recovery by persuading him to refrain from further ingestion of food.

Headache may occur in plethora, or it may occur in anemia. It may arise from increase of intracranial tension, or it may be provoked by a fall in intracranial tension, as after spinal puncture; though Gordon Holmes (quoted by Critchley and Ferguson) tells us that he has never known migraine to persist in a patient who had had a surgical or traumatic decompression. Headache may arise from excess of food in the gastrointestinal tract, or from an insufficiency. It is a symptom of most fevers, although I doubt whether anyone has ever successfully explained why. To the ophthalmic physician it is significant as a product of eyestrain, yet many patients with plenty of cause for eye-

strain proudly allege that they have never had a headache, do not know what headache means.

Opinion as to the exact seat of the pain in headache is almost as indefinite today as in the time of Galen (131-201 A.D.). In 1893, the question was reviewed historically by Harry Campbell of London (The Lancet, 1893, volume 2, page 184), who described headache as pain felt anywhere above the base of the skull.

Campbell argued that the aching structure might be "(1) the brain itself; (2) its membranes — the pia mater, arachnoid and dura mater — these including the tentorium cerebelli, the falx cerebri and cerebelli and the large fold of pia mater forming the velum interpositum; (3) the skull-bones and mucous membranes lining the frontal, ethmoid, sphenoid and mastoid sinuses and the middle ear; and, finally, (4) the structures covering the cranial vault, including the scalp, the skin of the forehead and temples and all the tissues lying between these and the bone — viz., the pericranium and certain aponeuroses and muscles."

Since it is at best exceptional for a patient to be able to refer his condition to any one of the structures just listed, his testimony as to the seat of the pain is obviously unreliable or useless.

The ancients agreed with modern medical thought in excluding the brain as a seat of pain. Hippocrates seems to have expressed no definite opinion on the question. Galen's "highly speculative mind" thought the arteries and brain capable of sensibility, but looked upon the meninges and pericranium as the principal seats of headache.

It is interesting to note that Galen distinguishes external headache from "internal pain by the fact that the internal pain spreads to the roots of the eyes (that is, the optic nerves), while the external one never does." His explanation of this supposed difference is that the tunics of the eyeballs are continuous with the cerebral meninges, such continuity facilitating transmission of the pain. This view was repeated, almost in Galen's own language, by writers of the sixteenth, seventeenth and eighteenth centuries. Galen placed the pain, in some cases of migraine, in the temporal muscles.

Willis (1621-1675), to whom medical science acknowledges a large indebtedness for fundamental knowledge in nervous anatomy and physiology, and who believed that pain depended upon a "convulsion or corrugation of the nerves," declares that the

parts affected by the pain of headache are "the two meninges and their various processes, the coats of the nerves, the pericranium . . . and other thin skinny membranes, the fleshy panicle of the muscle, and, lastly, the skin itself."

Seller (quoted by Campbell) mentions an opinion that the brain is devoid of sensation during health, but capable of becoming sensitive under certain morbid conditions, the sensibility being independent of nerves and altogether sui generis. Heberden (1710-1801), in his Commentaries, declares that headaches appear to be seated in the brain itself.

Gowers (1845-1915), master neurologist and ophthalmoscopist, made the interesting clinical observation that hemiplegia due to syphilitic thrombosis was very frequently (in one-half of his cases) preceded for days or even months by headache, and he remarks that the pain in these cases is "apparently in some way due to the arterial disease itself"; but he does not commit himself to the opinion that it is actually located in the arteries.

As already hinted, the importance of headache for the ophthalmologist lies in the fact that a large proportion of his patients come to him for relief from that particular symptom. Estimates as to the percentage of cases of headache wholly or partly due to eyestrain have varied from fifty to ninety or more. Headache is common even in young children. Many years ago Treichler stated that one-third of the children attending school in France and Germany suffered from headache.

Headaches attributed to eyestrain are sometimes due to entirely different causes, and it is equally true that headaches attributed to other factors are often relieved by correction of eyestrain. Thus, eye physicians must be prepared to arrive at a preliminary decision as to whether the headache which the patient describes may be or probably is due to eyestrain; but the eye physician must also know enough about other possible causes of headache to be able to suspect such causes either as acting independently and being responsible for a headache in which eyestrain has no part, or, perhaps more frequently, as conspiring with eyestrain to produce the symptomatic result.

For it must not be forgotten that headache, like some other symptoms, is more likely to occur in the presence of several causes than in the presence of only one cause.

Attempts are often made to decide whether or not we are dealing with an eyestrain headache by its location or by the time

of day at which the headache occurs. As very characteristic of eyestrain we include especially frontal and parietal headache or an ache occurring in and around the eyeball itself. But occipital and nuchal pains are frequently symptoms of eyestrain, and, at times, eyestrain leads the patient to complain of pain as low down as between the shoulder blades, which, of course, can hardly be thought of as a headache and yet is closely related to it.

Usually headache is most typically attributable to eyestrain when it comes on during close use of the eyes, or during such distant use of the eyes as in driving, looking out of a train window, or watching a theatrical performance. A child may develop headache after an hour or two in school and especially during classes which demand close use of the eyes.

If a headache is experienced on rising in the morning, or even during the night, we are a little more apt to be skeptical as to its ocular relationship. Yet many of us have seen patients in whom nocturnal or early morning headache was definitely traceable to strain put upon the eyes during the late hours of the previous day.

The oculist and the otolaryngologist often play a sort of game of "I spy," in which now one scores and now the other. The patient who has been fitted with several pairs of glasses, all pretty much alike, may some day drop in on his oculist with the remark, "Dr. Rhino cured my headache right off by washing out my antrum." A week later, the oculist may score by giving prompt relief to a patient who has been subjected to nasal packs for a month or so. The great principle of the game, if you play it warily, is to preserve an open mind, and to know a good deal about the other fellow's specialty as well as your own.

Another specialty with which the ophthalmologist needs to have distinctly more than a nodding acquaintance is that of the neurologist. Otherwise, he might make the mistake of assuming that an encephalitis developing after German measles was merely an indication for prescribing glasses. It is significant that some of the greatest writers on ophthalmoscopy have been neurologists, and it is a pity that not every ophthalmologist's training has included at least several months in a neurological clinic or hospital.

The need of at least a year of intimate contact with the practice of general medicine before entering the restricted field

of ophthalmology becomes more evident when we remember that the ophthalmologist may be the first physician consulted by a patient who is suffering from nephritis or intracranial tumor or one of the slower forms of meningitis. In a patient who has been under treatment for tuberculosis it is not always easy to arrive at a downright conclusion whether headache is due to general toxemia, eyestrain, or a meningeal complication. A patient's cerebral arteriosclerosis may have more — or less — to do with his discomfort than does the inadequacy of his refractive correction.

It is a very essential commonplace to say that we are often called upon to treat the patient rather than his symptoms. Headache may arise purely from general fatigue, although even more frequently from general fatigue in combination with eyestrain. It may not be amiss here to remind non-ophthalmologists that excellent vision does not demonstrate freedom from eyestrain, since the worst sufferers from headache often show so-called normal visual acuity without glasses and are relieved by a hyperopic or astigmatic correction which does not materially change their visual power.

Quite commonly we are taught that the myopic or short-sighted patient does not experience headache. There is some truth in this, but also some error. I have found a number of slightly myopic patients whose headaches were relieved upon correction of the myopia. I have no completely satisfactory explanation for this manifestation. In the presence of a low myopia affecting equally both eyes, use of the accommodation can hardly be a factor. But many of these patients resort to contraction of the muscles of the eyelids in the attempt to sharpen distant vision, and this I believe explains the headache or other symptoms of strain which they experience. A myopic patient may, of course, be subjected to eyestrain if his two eyes are unequal in focus or if the myopia is complicated by appreciable astigmatism.

The refractionist who habitually undercorrects hyperopia needs to be reminded that many headache victims can only obtain relief by patiently learning to wear a full correction, and that the full correction, even as found under cycloplegia, is most emphatically called for in this type of patient.

Headaches and some more remote symptoms of eyestrain bring us into the interesting field of referred pain. A patient with uncorrected hyperopia or astigmatism, or with an improper correction, may be subject to frequent attacks of so-called "in-

digestion" — nausea and even vomiting, often but not always in association with headache. Other referred or reflex symptoms which may occur in association with headache or may occur independently are dizziness, loss of the power of concentration, and various queer head sensations which each patient describes in his own peculiar way.

As Tilley has reminded us (British Medical Journal, 1933, volume 1, page 549), nerve sensations affecting the face and scalp are reflected into the gasserian ganglion and the pons, to be further distributed over an extensive area of brain stem from the level of the corpora quadrigemina to the upper cervical region of the spinal cord; and these nerve impulses find numerous interrelationships with the nuclei of origin of all the other cranial nerves. It must be equally true that nerve impulses from these remote regions are occasionally reflected to the head. The interrelationship between the head and face and the digestive apparatus is particularly intimate, because the sensory root of the fifth nerve lies in close approximation to the root of the vagus nerve in the floor of the fourth ventricle.

Headache as a symptom arising from disease of the nasal accessory sinuses has already been dealt with by Dr. Vail. In the interpretation of headache, the importance of the nasal sinuses for the ophthalmologist lies in the facts that they may produce headache, that they may even produce a headache which simulates ocular headache, that a headache thought to be due to the sinuses may actually be ocular, and that both the eye and the nasal sinuses may share the responsibility for headache.

When the ophthalmologist has done his best in the correction of a refractive error which seems important, the patient's failure to obtain relief may be due to faulty habits in reading. In many patients, excessively rapid reading is productive of cerebral fatigue which may translate itself into general exhaustion and inhibition of digestive function. In children, a sincere attempt to study home work in the evening in spite of distraction by the radio and other disturbances may cause headache and brain fag besides interfering with the intellectual results. On the other hand, steady deliberate work without distractions is not infrequently followed by a sense of restfulness and mental repose. The range or angle at which the book is placed before the eyes, the relative height of the patient and of the table or desk upon which a book is placed or writing is carried on, the question

whether study or reading too promptly follows ingestion of a heavy meal, may all have to be considered as supplemental factors.

Headache may arise from irregularities in diet or disorders of digestion. In this connection we may find ourselves confronted with problems relating to chronic gastrointestinal disease, of excessive intake of food, of allergic reactions, or of miscellaneous dietetic indiscretions. The question whether the patient partakes injuriously of alcohol or tobacco must not be overlooked.

Every physician, including the eye physician, must remember the importance of fatigue in producing digestive disturbances. In his classical observations on the stomach of Alexis St. Martin through a traumatic fistula, Beaumont records that in "fear, anger, or whatever depresses or disturbs the nervous system, the villous coat of the stomach becomes sometimes red and dry, at other times pale and moist, and loses its smooth and healthy appearance; the secretions become vitiated, greatly diminished, or entirely suppressed." On the other hand a regurgitation of bile into the stomach was precipitated when St. Martin became very angry. Alvarez suggests that just as sensitive persons blanch and blush externally, so also they may perhaps blanch and blush internally.

Many essays, and a few more extensive monographs, have been written on the subject of headache. Some of these have assembled interesting examples of the influence of headache upon the careers of scientists, artists, and distinguished authors.

It seems probable that Charles Darwin was a victim of uncorrected eyestrain, although the exhaustion which he experienced after facing an audience may have been largely emotional.

Sir Isaac Newton was compelled by headache to abandon his laborious studies on the theory of lunar irregularities; and he states that "his head had never ached but when studying that subject" (quoted in a monograph by Wright on headache). In this case the pain may have been due merely to excessive use of a brain which was threatened with disease.

One of the most famous of English literary critics of the last century, and perhaps the greatest student of the Renaissance period of Italian art, was John Addington Symonds the younger, clearly a victim of chronic tuberculosis, and ultimately dying from the complications of that disease, who suffered greatly from

headache and other symptoms in which eyestrain probably played an important part. He is the subject of one of George M. Gould's "Biographic Clinics." Gould, who indignantly rejected the title of "crank," shows his usual extravagance of viewpoint by attributing Symonds' tuberculosis to his eyestrain.

It may be that the younger Symonds' headaches, which were treated for some time by his father, John Addington Symonds, Sr., had something to do with the fact that the latter selected headache as the subject of an excellent monograph which he delivered before the Royal College of Physicians as the Gulstonian Lecture for 1858 (Medical Times and Gazette, 1858, volume 16). Perhaps the elder Symonds has his son's obstinate affliction in mind when he says: "Put all the breast-pains, stomach-pains and colic-pains together, and you do not make such an aggregate of suffering as is furnished by headache"; and again when he describes the common headache as "that torment to the individual and pest of society, which so often interrupts the daily occupation, spoils the arrangements of social life, and drives the sufferer to the domestic medicine chest, and thence to the family practitioner and the medical celebrities of the day; while but too often, from their baffled science and art, the patient flies frantic with disappointment to a series of quackeries, ending in greater disappointment, because beginning with more extravagant expectation." He mentions two cases in which headache was relieved by removal of teeth. Probably he again had his son in mind when he described nervous headaches as occurring in "persons of very lively emotions and delicate sensibility . . . among those who have the æsthetical and imaginative elements highly developed, . . . the frequent accompaniment and curse of high intellectual endowments," especially "when the functional activity of the brain, whether in perception, emotion, or intellect, is disproportionate to the organic vigour of the rest of the body."

There can be no question that many lives have been ruined by manifestations of uncorrected eyestrain, especially in the form of headache. Too often this fact is overlooked by careless refractionists. George M. Gould, on the other hand, allowed himself to be carried away by an excess of enthusiasm. In his Biographic Clinics (published over a period of seven years) he traced to eyestrain, frequently with headache as a leading symptom, the ill health of De Quincey, Carlyle, Charles Darwin, Thomas Huxley, Robert Browning, John Addington Symonds, George

Eliot, Herbert Spencer, John Greenleaf Whittier, Hippolyte Taine, Honoré de Balzac, Tchaikovsky, Flaubert, Jonathan Swift, and others. These essays are readable and suggestive, but must be accepted with the proverbial grain of salt.

It was Gould who at first estimated that ninety to ninety-five per cent of cases of migraine were due to eyestrain and correctable with glasses, and who subsequently raised this estimate to ninety-nine per cent!

As regards migraine, a wiser conclusion is represented by the opinion of a group of leaders in refraction, quoted by Gould, who assented to the following: "I believe that sick headache often depends on eyestrain, and I have known cases that were cured by ocular treatment."

When every possible consideration as to etiology has been explored and every therapeutic measure tried, it is likely that as regards many of its victims migraine will still remain a mystery, will still present unconquerable barriers. In the words of Critchley and Ferguson (The Lancet, 1933, volume 1, pp. 123 and 182), migraine "has been the happy hunting ground of the theorist, and the problem has been attacked by representatives of all branches of medicine. Each in turn has discovered in migraine phenomena pertaining to his own specialty; each in turn has hit upon the true nature of the malady; in turn, each has found the infallible remedy."

The ophthalmic physician must at all times be willing to cooperate to the utmost in the attempt to relieve the migraine subject: but he must also recognize that the cause may lie in other fields than his own, and that cure may be impossible.

Whereas migraine is ordinarily regarded as "a paroxysmal affection comprising severe unilateral headache, preceded by visual phenomena and followed by nausea and vomiting," Critchley and Ferguson argue that any one or even any two of these classical symptoms may be absent and the diagnosis of migraine still hold good. Thus, according to these authors, the essential disorder may be manifested solely by the disturbance of vision (zig-zag vision, scintillating scotoma, teichopsia), by the occurrence of paroxysmal headache, or by nausea and vomiting. Usually unilateral, the headache may lack this characteristic and still be migrainous in origin.

In recent years, an interesting addition to the possible causes of migraine, and also of other forms of headache, has been offered by the allergists, and the possibility of allergy as a basis for the disorder must be frankly recognized by the progressive ophthalmologist. It may be true, as Critchley and Ferguson declare, and as Alvarez is evidently also disposed to consider, that the case for the allergic nature of migraine has been overstated. Yet there is most convincing evidence that in some cases, at least, the migrainous attack is provoked by reaction to a foreign protein.

Balyeat and Brittain (American Journal of the Medical Sciences, 1930, volume 180, page 212) go so far as to say that the exciting factor in migraine is probably always a specific sensitivity to one or more foreign proteins, although there may be many predisposing factors.

In its bearing on the complexity of the problem of allergic migraine, the following comment by Balyeat and Brittain seems worthy of reproduction here: "Practically all investigators give heredity as the most potent factor in the production of migraine. It need not be assumed, as is frequently believed, that the hereditary factor must be migraine itself. . . . . It is only one of a number of symptom complexes which are metamorphosed in passing from parent to offspring and arise in different forms in different members of a family. For example, five children in one family might be specifically sensitive to wheat. From such a sensitivity asthmatic symptoms might appear in the first child, eczema in the second, hay fever in the third, urticaria in the fourth, and migraine in the fifth. . . . . The type of sensitivity from which the antecedent suffers has no relation to the type the descendent may have." In the series of migraine patients studied by Balyeat and Brittain, a positive family history of allergy was elicited in forty-seven, or 85.4 per cent, while there was a family history of migraine in only twenty-five, or 45.4 per cent.

Rowe (California and Western Medicine, 1930, volume 33, page 785), who feels that every case of migraine should be studied with the possibility of food allergy in mind, makes the following observations: "Many discussions of migraine, even today, lay most emphasis on neurosis as a cause. It is true that many of these sufferers are introspective, analytical, and neurotic. This is due in large measure, however, to their continued effort at self-help since all types of medical treatment have been of little benefit in the past."

Rowe believes that migraine resulting from food allergy is "due to a localized swelling or vascular spasm in the brain. Pain in some cases in the literature," he says, "has been so severe as to suggest brain tumor, and exploration has revealed angioneurotic edema of the brain itself. . . . . Such localized edemas would explain the transient amblyopia, aphasia, paresis, or paresthesia which have been described as complications of migraine for many decades."

Unfortunately, investigation as to substances to which the patient is allergic is expensive and often lacks finality. In addition to the relatively simple scratch tests, intradermal and patch tests and the use of elimination diets are recommended. Quoting from Hinnant (Cleveland Clinic Quarterly, 1935, volume 2, page 71): "Probably the laboratory procedure of greatest value in cases of food allergy is the leucopenic index of Vaughan which is based on the principle of hemoclastic crisis as pointed out by Widal, Abrami and Iancovesco in 1920. The ingestion of foods in normal patients produces a mild leucocytosis. In patients with food allergy, the ingestion of foods to which the patient is sensitive produces a marked leucopenia. Through this simple laboratory procedure, offending foods have been discovered when skin tests, elimination diets, and the food diary have failed. In the experience of Vaughan and other workers, this test has been from 90 to 100 per cent accurate."

Although nervous headaches may occur in people whom we do not ordinarily regard as nervous, the victims are often of the type which expends nerve energy excessively in the course of the routine of even a normal day.

When we talk of emotions and their disturbing influence upon bodily functions, we are a little too apt to think exclusively of the major and more obvious emotions of anger, fear, grief, or even joy. But many excellent people, in the course of an ordinary psychologic day, go through a multitude of petty emotional upsets — irritation over what someone else or even the individual himself has done, humiliating incidents hardly recognized as such at the moment and later forgotten, disappointment, eagerness, apprehension, a sense of the irksomeness of duties uncompleted, perplexity, suspense, the tension of responsibility, difficulty of concentration in the presence of distractions. It may be said that some persons live almost continually in a state of suppressed excitement, and the possibilities for resulting fatigue of the whole nervous system are readily understood.

The inveterate kindliness of Dr. Oliver Wendell Holmes, displayed alike in his miscellaneous writings and in his medical essays, stands in contrast with his frank recognition of the type of hopeless patient described in the following quotation from his valedictory address, in 1871, to the graduating class of Bellevue Hospital College: "What I call a good patient is one who, having found a good physician, sticks to him till he dies. But there are many very good people who are not what I call good patients. I was once requested to call on a lady suffering from nervous and other symptoms. It came out in the preliminary conversational skirmish, half medical, half social, that I was the twenty-sixth member of the faculty into whose arms, professionally speaking, she had successively thrown herself. Not being a believer in such a rapid rotation of scientific crops, I gently deposited the burden, commending it to the care of number twenty-seven, and him, whoever he might be, to the care of Heaven."

In conclusion, it may be said that, while the ophthalmologist must neither belittle nor neglect his own possibilities for helpfulness, and must at the same time recognize the variety of possibilities of cooperation by other physicians, he must in the end be also ready to accept the fact that there are patients who cannot be cured, because it is impossible to obtain their cooperation, either from lack of will or from sheer lack of the necessary mental balance in the patient himself.

SYMPOSIUM ON HEADACHE

* * *

C. FROM THE STANDPOINT OF THE NEUROLOGIST

A. R. VONDERAHE, M.D.*
BY INVITATION
CINCINNATI

I. Pain in the Head from Direct Irritation of Sensory Nerves

OF ALL human experiences, pain has perhaps the greatest reality, and even superficial observation connects it with anatomic changes. It is the purpose of this paper to inquire into the anatomic substratum of pain in the head region. Included here are the fairly localized pains of neuralgia and neuritis and the more diffuse pains, reflex in character, usually termed "headache." Pain in the head, accordingly, may be considered as arising in two general ways: (1) from direct irritation of sensory nerves transmitting pain impulses; (2) as reflex pains arising from an irritation somewhere else in the body. The sensory components of the cranial nerves are first considered.

The olfactory nerve, so far as is known, is concerned only with the special sense of smell. Care is taken to test this nerve clinically with aromatic substances such as oil of wintergreen or peppermint and not with irritating substances such as ammonia which would cause pain through irritation of that portion of the mucous membrane innervated by the trigeminal nerve.

The optic nerve, similarly, is concerned only with the special sense of vision. Pain arising from unusually strong visual stimuli is of the reflex type. The sheath of the optic nerve is innervated from filaments of the trigeminal nerve and may, when irritated, give rise to local pain. Occipital and frontal pain associated with refractive error is reflex in character with a referring of the pain to the trigeminal fibers of the dura mater in these areas. Occipital pain often occurs in cases where the

* From the Department of Anatomy (Neurology), University of Cincinnati Medical School.

patient strains the posterior neck muscles in compensating for diplopia; here the pain arises within the muscles themselves.

The eye muscle nerves, oculomotor, trochlear and abducens, have sensory components and the probability of local pain arising in their fibers is strongly present. The sensory fibers are surely proprioceptive, but evidence exists by analogy with nerves to muscles elsewhere in the body, that they also carry deep pain sense. Sensory ganglion cells associated with the oculomotor and abducens nerves were first noticed by Thomsen[1] in 1887, and confirmed by Nicholson[2] in 1924, while the sensory nature was further substantiated by the work of Sherrington and Tozer.[3] It is altogether possible to have local pain in the external eye muscles from fatigue arising from refractive error, overwork, or strain from any cause. In addition, of course, more diffuse headache of a reflex type may occur. The central representation of this afferent system includes the mesencephalic root and nucleus of the trigeminal nerve for proprioceptive sensation and possibly the main sensory nucleus of the trigeminal for pain sensation.

The trigeminal nerve is perhaps the most important of the sensory nerves of the head and an understanding of its distribution and characteristics is essential to an anatomic analysis of head pain. It is a common error, however, to associate all varieties of neuralgic pain over the anterior part of the head with this nerve: in this matter much chagrin has been caused to the physician by the persistence of pain in some cases after complete evulsion of the trigeminal root from the brain stem. Through its three divisions and their numerous important branches and connections it is a frequent source of direct painful irritation. In addition, it is the site of numerous pains which are reflex in character.

The ophthalmic nerve through its branches may be responsible for pain in the dura mater of the tentorium of the cerebellum, the region of the upper eyelid and lacrimal gland, the inner and lower part of the forehead, the frontal sinus, and the scalp and pericranium as far as the occipital bone. Further possibilities arise from the innervation of the ciliary ganglion and from its sensory supply to the muscles of the iris, ciliary muscle and cornea.

The maxillary nerve through its branches transmits pain impulses from the dura mater of the middle cranial fossa, the skin of the anterior temporal region, conjunctivæ and skin of

the lower eyelid, skin of the side of the face, anterior portion of the skin over the cheek, the mucous membrane and skin of the upper lip, as well as from the upper teeth. The sphenopalatine ganglion, associated with this nerve, presents an anatomic factor of special interest. Through this ganglion pass sensory fibers from the maxillary nerve and thoracolumbar sympathetic fibers of the deep petrosal nerve from the carotid plexus, while craniosacral (vagal) sympathetic fibers from the facial cranial nerve (transmitted by the greater superficial petrosal nerve) synapse with the intrinsic cells of the ganglion. Before entrance into the ganglion the two petrosal nerves, carrying contrasting sympathetic motor impulses, fuse to form the vidian nerve. The sphenopalatine ganglion transmits trigeminal pain fibers to the sphenoid and posterior ethmoid cells, the periosteum of the occiput, the mucous membranes of the posterior and superior part of the nasal fossa, the roof and septum of the nose, and the hard palate posterior to the incisor teeth.

The mandibular nerve opens up the possibility of pain in the skin on the side of the head, the auricle, external acoustic meatus, the lower portion of the face and lower lip, mucous membranes of the mouth, tongue, the ethmoid cells, lower teeth and gums, the salivary glands, temporomandibular articulation, and skin and scalp over the temporal region as far as the sagittal suture. The dura mater in the general distribution of the middle meningeal artery, an area supplied by this nerve, is the most frequent localization of pain in migraine of the hemicranial type. The possibility of deep pain sensation arising from the muscles of mastication and other muscles supplied by the motor division of the trigeminal nerve must also be considered. Associated with the mandibular nerve are the otic and submaxillary ganglia. The otic ganglion, transmitting sensory fibers from this nerve, sympathetic motor fibers from the plexus about the middle meningeal artery, and craniosacral sympathetic fibers from the lesser superficial petrosal nerve of the glossopharyngeal, destined for the parotid gland; and the submaxillary ganglion, transmitting sensory fibers from the mandibular nerve, parasympathetic fibers from the facial nerve and sympathetic fibers from the plexus surrounding the facial artery, to the sublingual and submaxillary glands, present possibilities of painful afferent stimulation analogous to those presented by the sphenopalatine ganglion.

In rare instances, pain may be set up by direct irritation of the main sensory nucleus and the descending root and nucleus

of the trigeminal nerve in the pons and medulla. In all cases of trigeminal neuralgia this possibility requires consideration and indicates the necessity for a complete neurological examination, since a lesion of the brain stem will produce characteristic neighborhood signs indicative of its central localization. Spiller[4] discusses this phenomenon in a case of syringomyelia. Weisenburg and Stack[5] discuss similar phenomena in a case of tuberculoma of the pons. Localized meningitis at the base of the brain in the region of the trigeminal root, tumors and other pathological involvements in this area, and tortuosity of the basilar artery sufficiently marked to cause pressure on the sensory root may all give rise to severe pains in the distribution of the trigeminal nerve.

The seventh cranial or facial nerve transmits special sensation of taste from the anterior two-thirds of the tongue, secretory fibers as just noted to the submaxillary and sublingual glands, and motor fibers to the muscles of expression. The transmission of deep pain sensibility over the so-called motor division of this nerve has been demonstrated by the work of Loyal Davis.[6] The possibility of pain arising in this nerve is readily overlooked. As a result of experimental work on animals and an analysis of clinical cases in which the trigeminal influences were removed by neurectomy Davis demonstrated the presence of intact deep pressure pain sensibility, and showed that the cells of origin of these pain fibers lie among the cells of the geniculate ganglion. It is important, therefore, in the analysis of neuralgia of the face to include among the possibilities pain arising from the facial nerve.

The eighth cranial nerve presents two special sensory functions, the special sense of hearing and special proprioceptive sense. So far as is known no pain sensation is transmitted over the auditory or vestibular nerves, however, unusual irritation of the acoustic or of the vestibular end organs is capable of producing reflex pains.

The glossopharyngeal nerve contains visceral afferent fibers. Some of these synapse with cells of the otic ganglion and proceed to the parotid gland. Others proceed to the mucous membranes of the middle ear, fauces, tongue, and pharynx; pain may arise directly from this system. Associated with the glossopharyngeal nerve is the superior ganglion and the petrosal ganglion. Some fibers emerging from the petrosal ganglion form the smaller superficial petrosal nerve which transmits sensory impulses and

some craniosacral impulses to the otic ganglion. Other fibers intermingle with those of vagus to supply the tongue and pharynx.

The vagus nerve distributes sensory fibers to the muscles of the soft palate, to the pharynx, larynx and to the abdominal and thoracic viscera. Its meningeal branch may be responsible for pain in the dura mater over the posterior fossa of the cranium. The auricular branch of the vagus supplies similar sensation to the skin over the posterior part of the auricle and the posterior inferior portion of the external acoustic meatus. The accessory portion of the spinal accessory nerve is merely a portion of the vagus nerve and is distributed with the fibers of the vagus. The spinal portion of this nerve does not concern us here. The vagus nerve plays its chief rôle in the production of numerous reflex headaches. From irritated end organs in thoracic and abdominal visceral disease arise impulses which travel to the brain stem, where they may be reflected over branches of the trigeminal system producing characteristic localized pains, or they may be reflected in motor impulses to blood vessels of the meninges and reach expression in types of migraine.

The hypoglossal nerve so far as is known is concerned only with the motor innervation of the tongue musculature. By analogy, however, with the external ocular nerves, it is not improbable that some deep pain sensibility may be likewise transmitted over this nerve.

The inferior and posterior portions of the cranium, sites of frequent pain, are innervated chiefly by the great occipital nerve which has its spinal representation in the second cervical segment. Neuralgia of this nerve occurs and has presented a troublesome problem in diagnosis.

II. Reflex Pain in the Head

As noted in the previous discussion, the cranial nerves of special sensation, while incapable of producing local pain, are capable of producing reflex pain in some associated areas of the head. Characteristic pains, accordingly, occur with disturbances of the optic, olfactory, vestibular, acoustic and gustatory nerves. The general nature of reflex pain may be briefly noted. Henry Head[7] states the general law: "When a painful stimulus is applied to a part of low sensibility in close central connection with a part of much greater sensibility, the pain produced is felt in the part of higher sensibility." A distinction must be made be-

tween referred pain as understood in this general conception of Head wherein a pain may be perceived in another portion of the body without changes in the musculature or in the caliber of the blood vessel walls, and reflex pains of another type which appear to be associated with such changes. Head's observation yields a basis for the association of various headaches with pathological changes in the thoracic and abdominal viscera, thus, painful stimulation flowing into the six upper thoracic segments tends to give headache in the nasofrontal area; the seventh thoracic segment (which corresponds to a visceral disorder at the base of the lungs, the upper portion of the stomach, the left side of the heart including the mitral region) yields a temporal headache; the eighth thoracic segment is associated with headache in the anteroparietal area; the ninth thoracic segment, the parietal area; and the tenth thoracic segment, the occipital area. In general, Head sums up the matter: "The higher up the affected portion of the trunk, the more anterior the affected zone of the head."

The exact anatomical mechanism and pathways concerned here cannot be stated with exactness. It may be called to mind, however, that afferent fibers from the viscera accompany the vagus nerve to the medulla and that connecting pathways associate this sensory system with the trigeminal so that an anatomic ground-work does exist for the radiation of pain, arising from pathologic states elsewhere in the body, into definite portions of the distribution of the trigeminal nerve in the head.

Reflex pain associated with changes of the vascular caliber may next be considered. It is generally held, though opinion to the contrary exists, that the constriction of a blood vessel itself does not set up pain. The stimulation of nerve ends by anoxemia resulting from constriction is held by some to be responsible for the pain. Whatever the final explanation, it is certainly true that vasoconstriction is associated with pain. With intense dilatation of the blood vessels, likewise, pain results; this is similar to the pain caused by distension of any other viscus and appears to be due to direct stimulation of sensory endings. The intense headache associated with brain tumor and with other pathologic states accompanied by increased intracranial tension are best explained by stretching irritation of the sensory nerve endings in the dura mater. Some light is thrown on the mechanism of migraine by these considerations. Lumbar puncture done during the attack of migraine shows no consistent change of

the cerebrospinal fluid pressure. Examination of the eye grounds in some instances has shown a spasm of the retinal arteries. Observation at the time of craniotomy has shown arterial contraction. It appears most likely that the best working hypothesis of the migraine attack assumes an alteration of vascular caliber or a sequence of such alterations. The hemicrania of the migraine attack lends itself to explanation by alteration of the caliber of the middle meningeal artery, the scotomata with alteration of the retinal artery. The brilliant work of Cobb and his associates[8] has demonstrated the presence of vasomotor fibers to the meningeal and cerebral vessels. The pharmacologists have for many years noted that vasomotor dilator drugs, such as amyl-nitrite and glycerol-trinitrite, may produce a severe throbbing headache. The studies of Pool and Nason[9] with ergotamine tartrate show a constant constriction of the arteries of the dura mater when the drug was used, while the effects on the pial arteries are inconstant. Riley[10] and many others have held the theory that arterial spasm may be the important factor in the migraine attack. Whether there is a vasoconstriction or vasodilatation or a sequence of the two, it seems certain that the migraine attack is associated with alteration of the caliber of the meningeal and, occasionally, the cerebral vessels.

In headaches associated with high blood pressure, several factors already mentioned may enter: reflex headache of cardiac origin, increased intracranial tension or cerebral edema with stretching of the dura mater, and local vascular reflexes associated with arteriosclerotic changes.

There remains to be considered headaches associated with psychoneurosis. These headaches tend to occur in the frontal or occipital areas, sometimes in both. When a complete examination is made of such an individual there is usually found, in addition to many signs of autonomic imbalance, generalized increased tonicity of muscles. An explanation of many of these headaches may be found in a strong, painful contraction of the occipito-frontalis muscle. When one considers that this muscle is part of a system for the expression of emotional states it can readily be seen how it may be thrown into a state of contraction by prolonged anxiety, intense fear or depression. Other neurotic headaches involve changes in the caliber of the blood vessels. The anatomic mechanism in these latter cases is capable of formulation. The underlying emotional state involves a conscious perception implicating the cerebral cortex. Pathways from

here descend to the hypothalamic area where centers dominating the sympathetic and parasympathetic systems are located and where patterns of the external expression of emotion, both visceral and somatic, are determined. As a result, therefore, of emotional conflict, widespread muscle tension and specific craniosacral and thorocolumbar phenomena occur, including vascular changes in the head resulting in associated headaches.

Conclusion

The difficulty in attempting a clinical classification of headache is recognized and there has been suggested here an approach to routine study of a clinical case by means of fundamental neuroanatomical pathways and mechanisms.

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THE HYPERTENSIVE FUNDUS OCULI AFTER RESECTION OF THE SPLANCHNIC SYMPATHETICS

A PRELIMINARY REPORT

F. Bruce Fralick, M.S., M.D.,* and
Max M. Peet, A.M., M.D.†
BY INVITATION

ANN ARBOR

It is well established that the interruption of the sympathetic fibers to the upper and lower extremities in man brings about an increase in the circulation of blood to the respective part. It has been assumed that the same result will be obtained if the sympathetic supply to the choroidal vessels is interrupted. This has been applied in retinitis pigmentosa. However, resection of the superior cervical sympathetic ganglia has not given the results hoped for. A more recent application of surgery to the sympathetic nervous system is section of the splanchnic nerves and lower dorsal sympathetic chain for hypertension. From an ophthalmological as well as from the general medical point of view, the results in some cases are very surprising.

Various methods have been tried in different clinics to permanently reduce high blood pressure. These include operations directed to the adrenal gland as the chief source of a vasopressure substance and have consisted either in division of the nerves close to the adrenal or in resection of a portion of each adrenal or extirpation of one gland. Permanent results have been disappointing following these procedures. Other operations have been directed against the sympathetic nervous system, Adson[1]

* From the Department of Ophthalmic Surgery, University of Michigan, Ann Arbor.
† From the Department of Neurosurgery, University of Michigan, Ann Arbor.

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having sectioned the anterior roots from the sixth dorsal to the second lumbar and Craig[2] sectioned the splanchnics below the diaphragm, together with the rami from the first and second lumbar ganglia. In the neurosurgical clinic at the University Hospital, a somewhat different approach has been used and we feel that the results, on the whole, indicate that this is the best procedure. The operation consists in resection of the greater and lesser splanchnic nerves and of the lower dorsal sympathetic chain, including the 10th, 11th, and 12th ganglia, through a supra-diaphragmatic approach, after resection of a portion of the 11th rib. The operation has always been performed bilaterally. Three objects may be obtained by this procedure. First, and probably the most important, is the relief of renal vasoconstriction. Richards[3] has shown that stimulating the splanchnic nerves increases the aortic blood pressure with subsequent impairment of kidney function. Goldblatt[4] has maintained hypertension in dogs by producing constant renal vasoconstriction by means of clamps on the renal arteries. The results of our operations indicate that in a rather high percentage of patients, renal vasoconstriction must have been an important factor in the disease, possibly the principal etiological factor, since relief of this vasoconstriction by splanchnic section has been followed not only by a permanent and often marked drop in blood pressure, but by marked improvement in kidney function.

The second object hoped for is the prevention of excessive adrenalin secretion by preventing stimulation of the suprarenal glands from higher centers. Section of the splanchnics should eliminate excessive adrenalin secretion resulting from excitement, fear, anger, and physical exertion. While we do not believe that hypersecretion of the adrenals is the cause of hypertension, it is probably an important factor in the sudden increases in pressure noted under various stimuli.

The third object is the production of a great vascular bed incapable of vasoconstriction. Section of the splanchnic nerves should prevent vasomotor spasm in the stomach, small intestine, liver, spleen, and kidneys, no matter what outside stimuli might

2. Craig, W. M.: *Unilateral and Bilateral Resection of the Major and Minor Splanchnic Nerves*, Arch. Int. Med. 54:577 (Oct.) 1934.
3. Richards, A. N.: *The Beaumont Foundation Lectures*, Sec. No. 8, Williams & Wilkins Co. (Baltimore) p. 11, 1929.
4. Goldblatt, H., Lynch, J., Hanzol, R. F., and Summerville, W. W.: *Studies on Experimental Hypertension*, J. Exp. Med. 59:347 (March 1) 1934.

be acting. This large vascular bed will, therefore, act as a safety valve and when peripheral vasoconstriction occurs, which under normal circumstances would greatly increase general vascular pressure, the vessels of the abdomen will dilate still further and the general blood pressure be kept at a lower level.

Section of the splanchnic nerves and the lower dorsal sympathetic chain apparently has no effect upon the general sympathetic system, either directly or reflexly. This would account for the failure to relieve fundus angiospasm in many of the patients. The fact that the angiospasm does disappear in some is difficult to explain. We have no explanation for the improvement, at times very striking, noted in the fundus following this operation for hypertension, unless we assume that the actual cause of the vascular spasm has been inhibited or removed. There may be some chemical substance produced in the kidney as a result of the continued renal spasm which has an irritative action upon the autonomic system. Diminution or absence of this chemical substance following splanchnic section would then account for the marked improvement noted in the fundus. However, such an assumption is entirely theoretical. The same may be said for the relief of edema and the absorption of exudates which occurs even more rapidly in some patients than would be suspected from the amount of improvement in blood pressure.

The presence of marked fundus changes, including papilloedema, extensive retinal hemorrhages, cotton wool patches and lipoid deposits has not been found a contraindication for operation. In fact, some of the most brilliant results have been in patients showing these ocular findings. At present, the only contraindications are advanced age, that is, patients over fifty, serious cardiac damage, and a high non-protein nitrogen in the blood. Purely on theoretical grounds, we have not felt that patients over fifty offered as good a prognosis as those somewhat younger. However, excellent results were obtained in one patient, age fifty-three.

Bilateral resection of the greater and lesser splanchnic nerves and of the lower dorsal sympathetic chain has now been performed in over 90 patients. All of these had systolic pressure of 200 or over. Many of these patients have not gone a sufficient length of time to enable us to draw definite conclusions. However, 42 patients have been operated upon a sufficient length of time, varying from 5 to 22 months, to give us a reasonable estimation of the post-operative results. Of these first 42, there

was an operative mortality of 4.8 per cent, no improvement in 12 per cent, slight improvement in 26 per cent, moderate improvement in 24 per cent, marked improvement in 19 per cent, and apparently cured 14.4 per cent. Among those apparently cured were patients showing marked fundus changes. Their blood pressures have remained normal for many months and there has been a complete restoration of kidney function. Seventy-five per cent of the patients had cerebral symptoms and 90 per cent of these had relief of these symptoms.

The operative results are most inconsistent in that those patients who, from the presence of advanced retinitis, arterial sclerosis, impaired kidney function, etc., would be expected to be poor candidates for the splanchnic resection, in some cases have responded most satisfactorily. In the early stages of this operation it was expected that the fundus picture would offer us our best criterion as to operability. This idea was soon abandoned since our prognosis as taken from the fundus findings was as often wrong as right. In fact, three of the patients showing the most severe hypertensive neuroretinitis together with severe organic vascular changes gave the best responses to the operation, both visually and generally.

Those patients showing no appreciable fall in their blood pressure, as a result of the splanchnic resection, were not improved visually or objectively as far as the fundus findings were concerned except in one instance. We are not able to account for the marked improvement which occurred here, but submit the case for the record:

Female, age 44, having a pre-operative blood pressure of 240/160, a vision of 6/12 in each eye, and a fairly normal kidney function (urinary concentration ability 1.033, urea clearance 71 per cent). She presented a fundus picture of a severe hypertensive neuroretinitis consisting of a disc edema of 2 D, markedly contracted and sclerotic arteries, diffuse marked retinal edema, macular star, and a moderate number of retinal hemorrhages. Nine months after a bilateral splanchnic resection had been performed, there was no change in the blood pressure or kidney function, but the edema and hemorrhages in the fundi had disappeared, the macular star was partly absorbed, and the vision improved to 6/6 + in each eye.

The eighteen patients showing an appreciable drop in blood pressure were benefited ophthalmoscopically in only three in-

stances. No change in the character of the contracted retinal arteries was noted. In one there was no change in vision, but the edema of the disc and retina was markedly less and the lipoids nearly all absorbed fifteen months post-operatively. The other two cases presented marked improvement in vision, but in only one of these was there any appreciable change in the fundus picture. This latter patient will be described in detail and might be considered as showing the greatest improvement in this group:

Male, 33 years of age, having a blood pressure of 260/175, a marked impairment of kidney function, (urinary concentration ability of 1.019, urea clearance 24 per cent), and a vision of 1/60 OD and 3/60 OS, presented the fundus picture of severe hypertensive neuroretinitis consisting of moderate edema of the discs and early secondary optic atrophy, marked sclerosis with general and local contraction of the arteries and arteriovenous nicking, and marked edema and scattered retinal lipoids. A bilateral splanchnic resection was performed and 17 months later the blood pressure had not risen above 180/140, the kidney function had returned to normal (urinary concentration ability 1.029, urea clearance 93 per cent), the vision had improved to 5/60 OD and 5/9 OS so that he could resume his occupation as a truck driver. The edema of the discs and retina had entirely disappeared, leaving only atrophy of both. The retinal vessels were, if anything, more sclerotic than previously. This is a truly remarkable improvement and the patient would have been placed in the group of so-called cures were it not for his blood pressure, which is too high to be considered normal.

It is interesting to note that the pre-operative fundi of the five so-called cured cases were those of malignant hypertensive neuroretinitis with choking in two cases, moderately severe hypertensive neuroretinitis in two and slight general and local angiospasm in the fifth. Post-operatively, it was not possible to follow the fundus changes in two of the latter cases to include them in this report. In the two malignant and one of the severe cases the exudates, hemorrhages, and edema disappeared and the vision, where reduced, was markedly improved. The angiospasm was apparently not relieved entirely since considerable and variable sclerosis was present. In fact, the sclerosis seemed to be more severe some months later than pre-operatively. A brief résumé of the above three cases will more adequately illustrate the fundus changes resulting from the splanchnic resection:

CASE 1 — Male, age 29. Pre-operative blood pressure 260/180. Kidney function impaired (concentration ability 1.014, urea clearance 105 per cent). Vision W.C. 6/60 each eye. Fundi: Discs swollen 2 D, arteries showed generalized contraction with local angiospastic areas, slight sclerosis and some arteriovenous nicking, retina presented marked diffuse edema, many hemorrhages of various types, scattered lipoids and macular star. A bilateral splanchnic resection was performed and twenty-seven months later the blood pressure had not risen above 134/78. The kidney function had returned to normal (concentration ability 1.030, urea clearance 117 per cent). The vision had improved to 6/6 — 3 OD and 6/12 + 2 OS. Fundi showed only moderate arteriosclerosis with generalized contraction and moderate arteriovenous nicking. The retina showed no edema but gave evidence of the previous retinitis by the presence of glial scarring and a few small lipoids.

CASE 2 — Male, age 52. Pre-operative blood pressure 260/180. Kidney function impaired moderately (concentration ability 1.023, urea clearance 70 per cent). Vision OD 6/30, OS enucleated for secondary glaucoma following central retinal venous occlusion. Fundus: disc swollen 2 D, arteries generally contracted with local areas of angiospasm and moderate sclerosis, retina moderately and diffusely edematous with a marked number of hemorrhages and few lipoids. A bilateral splanchnic resection was performed and when last seen seven months postoperative the blood pressure was 166/108, the kidney function was improved but still subnormal (concentration ability 1.027, urea clearance 75 per cent). The vision had improved to 6/6 OD. The disc showed no edema or atrophy. Arteries were moderately sclerotic with generalized contraction and arteriovenous nicking. Retina was negative aside from a few small lipoids.

CASE 3 — Female, age 22. Pre-operative blood pressure 280/190. Kidney function normal (concentration ability 1.033, urea clearance 87 per cent). Vision 6/6 each eye. Fundi: Discs slightly edematous, and show one hemorrhagic area, arteries show marked variation in caliber, generally contracted and questionably slightly sclerotic, moderate retinal edema containing a few cotton wool patches and hemorrhages. A bilateral splanchnic resection was performed and eleven months later the blood pressure had not again risen over 132/95. The kidney function

was still normal (concentration ability 1.029, urea clearance 85 per cent). The vision was still 6/6 in each eye. Fundi: discs negative, arteries normal in size and uniform in caliber but showing slight sclerosis, retina normal aside from a few glial streaks.

Comments

The presence of a hypertensive retinitis is always of serious prognostic significance. It is, therefore, imperative that we recognize arterial constriction or angiospasm as the precursor of angiospastic retinitis and its more severe complications of organic vascular changes.

One of the most difficult problems encountered in the study of the fundi in this series of cases was our frequent inability to differentiate between angiospasm and early sclerosis of the retinal arteries. The former would be expected to respond to a lowering of the blood pressure, while the latter would remain more or less unchanged. The tendency to rapid disappearance of the retinitis after lowering of the blood pressure through splanchnic resection would indicate that they are the visible signs of an angiospastic rather than an arteriosclerotic lesion. Most of the cases observed were a combination of both; the organic vascular changes being a by-product of the angiospasm. The more severe the visible sclerosis, the more difficult was it to demonstrate areas of localized angiospasm. If we agree that in the areas of long-standing severe angiospasm, retinitis develops, then the localized areas of retinitis, consisting of edema and hemorrhages, must be recognized as being associated with local spasms of the retinal arteries even though masked by the sclerosis present. In the more severe forms of retinitis consisting of general angiospasm, generalized edema of the disc and retina, retinal lipoids, hemorrhages, cotton wool patches and hyperemia, a diagnosis of malignant hypertension is usually made. This is accompanied by a prognosis for life of from 18 to 24 months. Two of the so-called cured cases and one of the improved cases in this series fall into this group and they are the only examples of malignant hypertension observed by us that have been benefited by any previous form of therapy.

That such apparent cure has occurred in this hitherto unbenefited condition is of the utmost significance. The operation is directed towards the relief of angiospasm and is one step forward in our efforts to the discovery of the causes of the tendency to angiospasm itself.

SUMMARY

1. Over ninety cases of essential hypertension having a blood pressure of over 200 were subjected to bilateral splanchnic resection.

2. A résumé of the history and rationale of the operation is discussed together with a statement regarding the physiology of the splanchnic nerves.

3. The three objectives towards which the operative intervention is directed are, first, the relief of renal vasoconstriction; second, the prevention of excessive adrenalin secretion by preventing stimulation of the suprarenal glands from higher centers; and third, the production of a great vascular bed incapable of vasoconstriction.

4. No criterion as to operability has as yet been established. A high non-protein nitrogen is a definite contraindication and individuals over fifty do not ordinarily respond well.

5. Thirty-six patients have been checked from five to twenty-two months post-operatively.

6. Five are symptom free and maintain a normal blood pressure. Of these, two showed resolution of the fundus changes of malignant hypertension with marked improvement in vision. One showed a resolution of an angiospastic retinitis. Two cases in this group could not be followed ophthalmoscopically.

7. Eighteen cases showed an appreciable drop in blood pressure with general symptomatic improvement, but in only three of these cases were there signs of improvement in the fundi.

8. The thirteen patients receiving no lowering in their blood pressure showed no improvement in either their fundus picture or vision except in one striking and unexplained instance where both were markedly improved.

DISCUSSION

DR. A. D. RUEDEMANN, Cleveland: The paper by Fralick and Peet is timely and well done. The results, however, should not be presented on a percentage basis, because as they become more selective in their cases, more accurate in their ability to pick cases, the results will be better. It is my contention that anything that offers a hope to the person with high blood pressure is of value because the present-day method of convicting a man to complete leisure and slow death as an alternative to work and rapid death is of little or no value, because the man works and dies — the man who knows how to take it leisurely does not have high blood pressure.

My experience is similar to that of the authors' in a series of equal size. Dr. Crile, some years ago, started doing the so-called adrenal denervation operation for hypertension. The operation is a major one. He strips the adrenal nerve completely loose from the splanchnic system and in so doing denervates the adrenal gland, causing a rapid temporary drop in blood pressure, which may last as long as six months. Thirty of these patients have been operated to date, most of them at the patient's request and some at mine. The physical results were poor from the viewpoint of permanently lowering the pressure. The symptomatic results were better and many of the patients were relieved of the full feeling in the head and their headaches. The fundus picture was not changed sufficiently to be diagnostic. The operation does not prolong life or change the picture.

In a second group of nearly 40 patients who had a denervation operation plus a splanchnic resection, the symptomatic results were better and the blood pressure and other physical results definitely better, a fall of nearly 100 mm. in blood pressure being recorded in several cases.

In the series of Fralick and Peet, their cases had over 200 mm. Hg. blood pressure. I believe their results will be better when the patients are operated earlier, as blood pressure over 200 mm. Hg. constitutes a serious risk. It is also well to repeat that the diastolic pressure is probably more important, especially if it is high. Early surgery and younger patients are two essentials for good results, as older patients frequently have vascular changes of senility plus spasm and myocardial changes which are beyond repair.

My experience is in accord with that noted by Fralick that the fundus picture is misleading.

Personally, I have obtained considerable aid by slit-lamp observations on the conjunctival vessels. Hemorrhages are seen earlier in the capillaries and I feel that the capillaries are more important than the larger vessels in the cases in which good results from surgery are to be obtained — the so-called spastic cases. The capillaries can be studied as to number per field, rate of blood flow, type of flow, and later by their recovery in number, flow, and width.

Before splanchnic resection, the finer vessels are tortuous, serpentine in character, have pigtail tortuositis, and the larger vessels show a peculiar scalloping as the walls are heavy and twisted at certain points. These changes are lost in many instances. It is also true that in some patients there was no material difference in the appearance of the capillaries before and after operation.

Retinal or conjunctival hemorrhages are signs of vascular degeneration of value to the surgeon before operation, as during operation the pressure is apt to mount and a cerebral accident take place which can be avoided in most instances when the danger is known. A clear white sclera due to conjunctival atrophy with a few straight and narrow capillaries having a constant running flow of blood and that do not open with dionin is a definite contraindication to surgery. Patients having such changes in conjunctival vessels are not benefited symptomatically nor is there any effect on the blood pressure or other physical signs.

We have been using the conjunctiva as one would the histamine flare test on the arm. If the conjunctival vessels appear spastic or narrowed, a drop of dionin solution one-half per cent will give a reaction by which the vessels may be studied in regard to the number obliterated and the number still functioning.

The flare on the arm and the conjunctival reaction are similar, but one can observe the latter and see the vessels dilate and observe the total number of capillaries present that would function if allowed to open.

Because vascular, spasm and arteriosclerosis are difficult in some instances for one to differentiate. I believe we should use all the vessels and all the aids we can assemble. Many more observations are essential before we know their diagnostic value.

Further, I believe more effort should be devoted to the venous side of the system. This is not a negative system. It suffers the same toxic insults as does the arterial side. It frequently is affected first and the tortuosities are seen earlier even though the pressure is lower. Hemorrhages are fewer with the veins. In some instances, I have felt that the obstruction was venous as the arterial side seemed full and the vessels ample in number. Post-surgically, a number of these patients have developed generalized venous engorgement, early varicosities in the legs, and dilated temple veins.

Do not deny these patients surgery on your ability as a fundus diagnostician for some surgeon will operate and you may see hemorrhages, edema, and lipoid infiltration disappear like magic.

As Fralick has pointed out, some of these patients are symptomatically helped, although the blood pressure is not lowered to any extent. The condition of the vessel wall and the question whether any vascular degeneration had affected the kidney and whether the changes were permanent or not are very important.

The vascular system is still a great unknown field and, as ophthalmologists, we can take an active part in the progress of diagnosis.

This paper by Fralick is a step forward and more work similar in character is necessary to carry the work on.

DR. F. B. BLACKMAR, Columbus, Ga.: I would like to speak about the mechanism of this improvement that you have heard about today.

Dr. Fralick is apparently of the opinion that the dilatation following his operations improves the function of the suprarenal gland. I wonder if it may not be that this gland is exhausted and that by splanchnic denervation of the gland he allows this exhausted gland to build up again?

My reason for saying this is that I have been using adrenal cortex extract for these hypertensive cases. We started using this cortex extract for allergic patients. We came across some allergic patients in middle life who had high blood pressure, and we were afraid we might increase the blood pressure, so we began using it in minute doses, gradually increasing, for allergic symptoms. Their blood pressures began to fall instead of rise. Apparently, the blood pressures will come down to 120 and stay there. They don't go any lower.

I just want to suggest that difference of opinion.

DR. E. M. JOSEPHSON, New York: I asked to discuss this paper for the very reason that Dr. Blackmar asked to discuss it — the mechanism. I have reported in the literature the response of blood pressure to the adrenal cortex hormone, which he mentions. It is a medical record of last March.

In my work with the adrenal cortex hormone, I have had occasion to confirm in many respects this antagonism between the adrenal cortex and the adrenal medulla. If you will recall the bacteriology, you will recall the close hookup between the medulla and the sympathetic system.

Rather than support the hypothesis Dr. Blackmar has enunciated here, I would suggest that by splanchnic resection, in all probability, the adrenal medullary function is cut down.

DR. HENRY P. WAGENER, Rochester, Minn.: The neurologic surgeons at the Mayo Clinic have operated on a number of cases of hypertension of varying degrees of severity. In the majority of the cases the technique used has been laminectomy and rhizotomy of the anterior roots from the 6th dorsal to the 2nd lumbar. A certain number of splanchnic sympathectomies have been performed. Very definite reductions in blood pressure have been obtained in a majority of the cases.

In none of the patients observed has there been any demonstrable change in the degree of the organic or sclerotic lesions in the retinal arterioles. In one patient, a tonic spasm of a retinal arteriole was found to be relaxed immediately after the operation, possibly as a result of the prolonged general anesthesia.

Rhizotomy was performed on eight patients with retinitis of hypertension, group 4 (malignant hypertension with edema of the disks). The retinitis was unchanged in three, improved in three, and cleared up in one. One patient died a few days postoperatively. Three others died within six months after the operation.

Rhizotomy was performed on twelve patients with retinitis of hypertension, group 3 (retinitis without edema of the disks). The retinitis cleared up in two, was improved in four, was unchanged in five, and recurred shortly after operation in one. Two of these patients died within nine months after operation. The retinitis had not improved in these cases.

Rhizotomy was performed in seven patients with sclerosis but without retinitis. The vessel changes were unchanged in these cases, but retinitis occurred in one patient four years after operation.

No essential change has been observed in the fundus picture of five patients on whom splanchnic sympathectomy was performed. Three of these patients had retinitis of hypertension, group 3, and two sclerosis only. In one of the latter, a mild retinitis was found to be present ten days after the operation.

Complete disappearance of the retinitis has occurred in three of twenty-three patients (13 per cent) and improvement of the retinitis in seven (30 per cent). I am not at all sure that these figures are higher than those obtained by more conservative methods of treatment.

DR. F. BRUCE FRALICK, closing: I wish to thank the discussants for their constructive views on this topic of hypertension. The work which Dr. Ruedemann is pursuing in the line of studying the characteristics of the conjunctival vascular changes in hypertension will aid us in more fully understanding this complex problem. Many times have I wished for Dr. Ruedemann and his apparatus in studying the changes following splanchnic sympathectomy.

Dr. Wagener has clarified to a considerable extent the ophthalmologist's conception of hypertension in relation to the fundus changes noted, thereby correlating the two.

Just what the ultimate future has in store for those cases of our series on whom a splanchnic sympathetic nerve resection has been performed is hard to conjecture. Whether their lives will be thereby lengthened or shortened only time will tell. Few seem to be made more uncomfortable by the operation and many receive at least a symptomatic improvement, unobtainable by any of the other therapeutics used.

The operation is obviously not the whole answer to the problem of hypertension as evidenced by the variability in the results obtained and by the recent work of Pick* who found that if he removes three-fourths of a dog's kidney tissue, its blood pressure will become markedly elevated. If he then withdraw 20 cc. of this animal's blood and inject it into another dog, the second animal's blood pressure will also rise immediately and stay up for several days. Further than this, if he then inject into the second dog 20 cc. of blood from a third dog whose kidneys have simply been denervated, the second dog's blood pressure will then sink. This series of experiments is important in that it tends to show that an intact kidney delivers into the circulation some pressor substance not delivered by a denervated kidney, and second, that a denervated kidney elaborates a hormone which causes blood pressure to fall.

* Pick, E. P.: Wiener Klinische Wochenschrift, (June 18) 1935.

ROENTGENOGRAPHIC DIAGNOSIS OF RETINOBLASTOMA

RAYMOND L. PFEIFFER, M.S., M.D.
BY INVITATION
NEW YORK

THE DIAGNOSIS of retinoblastoma, or so-called glioma of the retina, is often made with some degree of mental reserve. Retinoblastomata may be closely simulated by other and benign conditions, and certain differentiation is sometimes impossible. Therefore, any aid, however limited in application, which will distinguish the real from the pseudo-tumor should be of interest.

It has long been known that calcareous changes or deposits occur in retinoblastomata. Three of the seven cases of glioma published by H. Knapp[1] in 1869 in his celebrated monograph on intraocular tumors, showed calcium bodies in histologic sections. Much earlier than this, Charles Robin[1] described calcareous degeneration in gliomata. These calcareous deposits appear in the histologic sections as irregular bodies or masses, 30 microns to a millimeter or more in size, which stain by hemotoxylin and are usually found associated with other degenerative changes in the tumor. According to Collins,[2] they are formed by the breaking down of a number of cells to form a granular mass in which lime salts are deposited and cholesterin crystals formed.

The calcium content of retinoblastomata was studied to determine its frequency and extent for possible adaptation to roentgenographic diagnosis. Roentgenograms were made of twenty enucleated eyes with retinoblastoma to determine the calcium content of the tumors, and subsequently twelve patients with eyes in situ with the clinical diagnosis of probable retinoblastoma were subjected to roentgenography of the orbits before operation.

ROENTGENOGRAPHY OF THE ENUCLEATED GLOBES

The roentgenograms of the enucleated eyes containing retinoblastoma showed fifteen of the twenty to contain calcium deposits. The specimens used were half globes, or less, from specimen

bottles. It is probable that several more would have revealed calcium had the whole tumors been available. For this reason also, the shadows of calcium on the films represent only fractions of the amount of calcium contained in the whole tumors. A comparative study of the individual specimens and their corresponding shadows on the films must likewise be qualified. The tumors which did not give shadows of calcium bodies, however, were the smallest of the series, while the largest and most advanced — that is, those in the second stage, — gave shadows of dense masses of calcium; and the various tumors of intermediate size and development gave shadows seemingly commensurate with their size and age. These shadows of calcium on the roentgenograms were composits of shadows of single psammous bodies which appeared faintly and widely dispersed in some, in small clumps in others, and in dense masses in the largest. (Fig. 1).

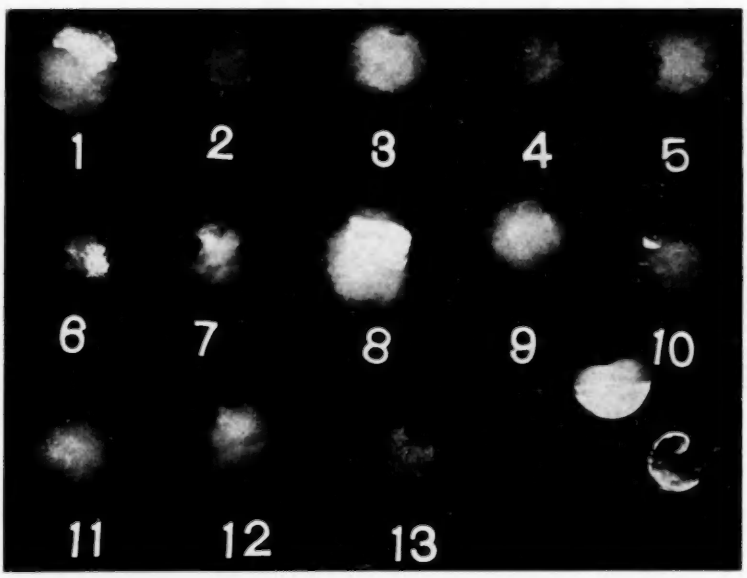

FIG. 1 — (Pfeiffer) — A roentgenogram of half-eyes from specimen bottles with thirteen retinoblastomata and three pseudotumors or phthisical eyes, loaned by Mr. E. B. Burchell of the New York Eye and Ear Infirmary. Ten of the thirteen eyes with tumors show calcium deposits of varying extent and density. Two of the three phthisical eyes in the lower right corner show shadows of calcium deposits, i.e., bone in the choroid in two, and calcium in a lens.

Calcareous degeneration is inclined to be patchy or localized in retinoblastomata, and unless sections are prepared and studied in series it is not possible to judge the amount of calcium present

in a tumor by the histologic method. A section may pass through the tumor and not through an area of degeneration, and thus may fail to reveal its presence. By using the roentgen ray the entire tumor or globe may be analyzed for calcium. Roentgenograms reveal the extent, position, and character of the calcium deposition, and may be made a laboratory adjunct to the pathologic study of retinoblastomata and atrophic or phthisical globes.

STUDY OF PATIENTS

Eight of the twelve patients with clinical diagnosis of probable retinoblastoma examined with the roentgen ray before operation were subsequently proved by histologic sections to have tumors, and are described below. Six of the eight revealed typical shadows of psammous bodies in the orbits before enucleation. Four of the patients had bilateral retinoblastomata, and three of these showed shadows in both orbits. Two retinoblastomata failed to show calcium in the orbits before enucleation, and when subsequently examined with the roentgen ray and in histologic sections were found to be entirely lacking in calcium. Four of the patients had pseudo-gliomata by histologic examination, and all failed to show calcium in the orbits when roentgenrayed before enucleation. One of the pseudogliomata, however, was found histologically to have a very small plaque of calcium in the organized tissue (Table I).

A ninth case of retinoblastoma is included with the cases presented because the tumor was small and the deposit of calcium dense and characteristic in nature. A drawing of the fundus is shown. The eye was not removed and histologic confirmation not obtained; thus, this case is not included in our statistics.

Case I (453023) — *Bilateral retinoblastomata, left, in second stage. Bilateral typical shadows of calcium deposits in orbital roentgenograms. Removal of left eye and demonstration of calcium in globe.* B. L., an infant girl of 17 months, was observed to have a whitish pupil in the left eye several weeks before examination. The left eye was slightly enlarged, tension elevated, and the pupil fixed. The right eye had normal tension and indirectly reactive pupil. Examination with anesthesia disclosed a large, yellowish-white, vascularized mass close to the lens in the left eye. The right eye contained a moderately large yellowish mass below the disc. Stereo roentgenograms showed typical granular shadows of calcareous degeneration in both tumors, a much larger

TABLE I — *Summary of the twelve patients with clinical diagnosis of retinoblastoma studied with aid of the roentgen ray before and after operation*

CHART NUMBER	X-RAY NUMBER	NAME	EYE	CLINICAL DIAGNOSIS	PATHOLOGICAL DIAGNOSIS	ROENTGENOGRAPHY OF ORBIT BEFORE ENUCLEATION	ROENTGENOGRAPHY OF GLOBE AFTER ENUCLEATION
453023	131751	B.L.	O.D.	Glioma		Typical calcium shadow	
			O.S.	Glioma	Glioma	Typical calcium shadow	Typical calcium shadow
440391	127232	A.G.	O.D.	Glioma		Indefinite shadow	
			O.S.	Glioma	Glioma	Typical calcium shadow	Typical calcium shadow
381958	109055	C.T.	O.D.	Glioma	Glioma	Typical calcium shadow	Typical calcium shadow
			O.S.	Glioma		Typical calcium shadow	
447179	131709	E.A.	O.D.	Glioma		No shadow	
			O.S.	Glioma	Glioma	Typical calcium shadow	Typical calcium shadow
461671	135342	M.F.	O.D.				
			O.S.	Glioma or pseudo	Glioma	Typical calcium shadow	Typical calcium shadow
421507	120478	G.R.	O.D.				
			O.S.	Glioma	Glioma	Typical calcium shadow	Typical calcium shadow
405166	116723	A.G.	O.D.	Glioma	Glioma	No shadow of calcium	No shadow of calcium
			O.S.				
430802	123762	M.S.	O.D.				
			O.S.	Glioma	Glioma	No shadow of calcium	No shadow of calcium
395004	106255	J.S.	O.D.	Glioma or pseudo	Pseudo	No shadow of calcium	No shadow of calcium
			O.S.				
354695	112782	H.M.	O.D.				
			O.S.	Glioma or pseudo	Pseudo	No shadow of calcium	No shadow of calcium
398875	116788	L.B.	O.D.				
			O.S.	Glioma or pseudo	Pseudo	No shadow of calcium	No shadow of calcium
421304	120464	R.A.	O.D.	Glioma or pseudo	Pseudo	No shadow of calcium	No shadow of calcium
			O.S.				

mass in the left. The left eye was removed, and roentgenograms were made to show the extent of the calcium deposition. Pathologic diagnosis: retinoblastoma. (Fig. 2).

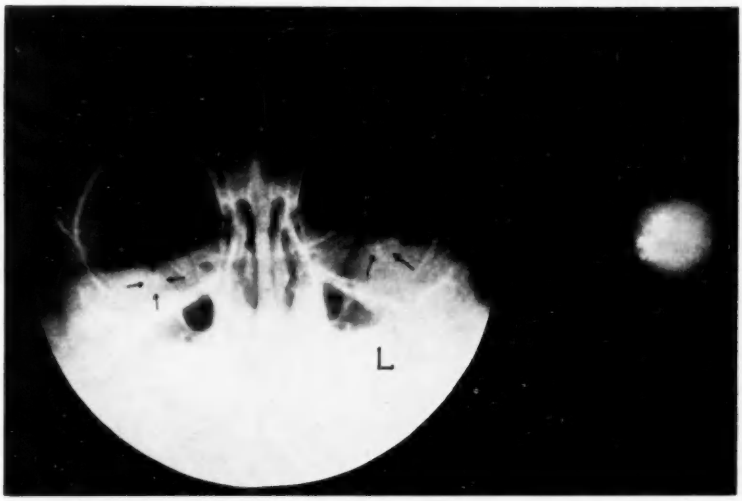

FIG. 2.—(Pfeiffer).—Roentgenograms of the orbits of the first case before, and of the left eye after enucleation. Note the granular character of the shadows of the psammous bodies. Stereoscopic films reveal much more clearly the deposits in the orbits.

CASE II (440391).—*Bilateral retinoblastomata, both in first stage. Bilateral shadows of calcium in roentgenograms of orbits. Enucleation of left eye and demonstration of calcium.* A. G., an infant girl of 14 months, was observed to have a whitish reflex from the left pupil one month earlier. Each eye had normal tension and normally reactive pupils. Examination of the child under anesthesia disclosed in the left eye a large tumor half filling the vitreous chamber. The right eye also revealed a retinoblastoma which was small and circumscribed, measuring 3 to 4 disc diameters. Stereo roentgenograms of the orbits revealed shadows of calcium granules in the position of the left globe and a minute and indefinite clump of shadows in the right. The left eye was removed and by the roentgen ray revealed a moderate sized mass of calcium. Pathologic diagnosis: retinoblastoma.

CASE III (381958).—*Bilateral retinoblastomata, the right in second stage. Typical shadow of calcium in right orbit in roentgenogram. Enucleation and demonstration of calcium. Eighteen months later, calcium revealed in left eye by the roentgen ray.*

C. T., a girl of two years, was observed to have a white pupil of the right eye five months earlier. This eye was slightly enlarged, the tension was elevated, the cornea steamy, and the pupil whitish. Examination under general anesthesia revealed a fairly large tumor mass in the left eye, also, which was in the first stage. Roentgenograms revealed a typical shadow of calcium deposition in the position of the right eye in the film of the orbits. The right eye was enucleated and roentgenograms of the globe revealed a moderately dense mass of calcium. Histologic diagnosis: retinoblastoma.

The left eye was treated with irradiation, and 18 months later roentgenograms of the orbits revealed a shadow of calcium in the position of the left eye.

Case IV (447179) — *Bilateral retinoblastomata, left in second stage. Demonstration of calcium in the left orbit. Enucleation and demonstration of calcareous degeneration after enucleation.* E. A., an infant boy of 11 months, was brought for diagnosis because of a whitish reflex from the left pupil, which was observed six months earlier. The left eye was enlarged and firm, the anterior chamber shallow, the iris fixed, and the pupil yellowish. The right eye contained three small yellowish, vascular, and circumscribed masses below the disc, had normal tension and a directly reactive pupil. Roentgenograms of the orbits showed the typical granular shadow of calcium degeneration of the tumor in the left eye, and a faint and indefinite shadow in the right. The left eye was enucleated and the calcium again demonstrated by the roentgen ray. Pathologic diagnosis: retinoblastoma. (Fig. 3).

Case V (461671) — *Unilateral retinoblastoma in second stage. Clinical appearance of pseudo-glioma. Demonstration of calcium and diagnosis of retinoblastoma. Enucleation.* M. F., a girl of four years, was observed to have "something wrong" with the left eye since the age of two. When tonsils were removed three months previously the surgeon advised that the left eye be examined. This eye showed a slightly hazy cornea, shallow anterior chamber, elevated tension and no enlargement. With the child under anesthesia the interior was indistinctly viewed. However, three widespread, flat, whitish areas could be observed, the largest below. The vitreous was clouded, and the surfaces of the whitish areas seemed soft and irregular. Stereo roentgenograms revealed a small, irregular shadow in the left orbit, apparently in the position of the largest mass observed in the

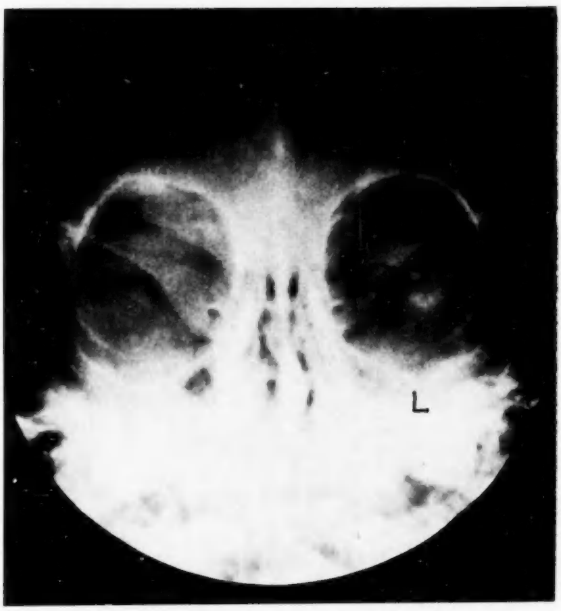

FIG. 3 — (Pfeiffer) — Roentgenograms of the orbits of the fourth case showing the dense deposit of calcium in the left globe.

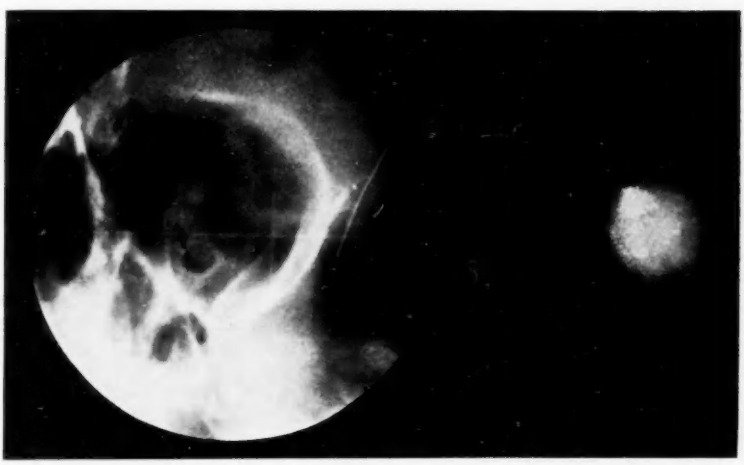

FIG. 4 — (Pfeiffer) — Roentgenograms of the sixth case before, and of the left eye after enucleation.

globe. The eye was removed and again roentgenographed to show the small mass of calcium. Pathologic diagnosis: retinoblastoma.

CASE VI (421507) — *Unilateral retinoblastoma in second stage. Demonstration of calcium before and after enucleation.* G. R., a female child of three years, was brought for examination because of a whitish pupil in the left eye which was observed six months earlier. The eye was large, the tension elevated, the anterior chamber was shallow, the iris fixed, and the pupil yellowish. Stereo roentgenograms of the left orbit revealed a fairly large shadow of calcium in the position of the eye. The eye was enucleated and again roentgenographed to show the large mass of calcium. Pathologic diagnosis: retinoblastoma. (Fig. 4).

CASE VII (405166) — *Retinoblastoma in first stage. Roentgenograms of orbits failed to show calcium. Enucleation. Roentgenograms of globe did not reveal calcium. No calcareous degeneration found in histologic sections.* A. G., a boy of two years, was brought for examination because a "white spot" was observed in the right eye a few weeks previously. Examination with anesthesia disclosed a yellowish mass in the temporal half of the globe with cell clusters projecting into the vitreous. Roentgenograms of the orbits failed to show shadows of calcium. The eye was enucleated and again roentgenographed, and was found to be negative for calcium. Histologic section failed to show calcareous degeneration. Pathologic diagnosis: retinoblastoma.

CASE VIII (430802) — *Unilateral retinoblastoma in first stage. Roentgenograms of orbits failed to show calcium. Enucleation. No calcareous degeneration in tumor.* M. S., a fourteen months old infant girl, was brought for examination because a detachment of the retina of the left eye was observed by a pediatrician. The tension of the eye was normal and the pupil reacted normally. The interior contained a large yellowish mass in the retina inferiorly, which extended nasally and temporally. Several floating clumps of cells were present in the vitreous. Roentgenograms of the orbits failed to show shadows of calcium deposits. The eye was enucleated and roentgenograms of the globe also failed to show calcium. Histologic sections likewise failed to show calcareous degeneration. Pathologic diagnosis: retinoblastoma.

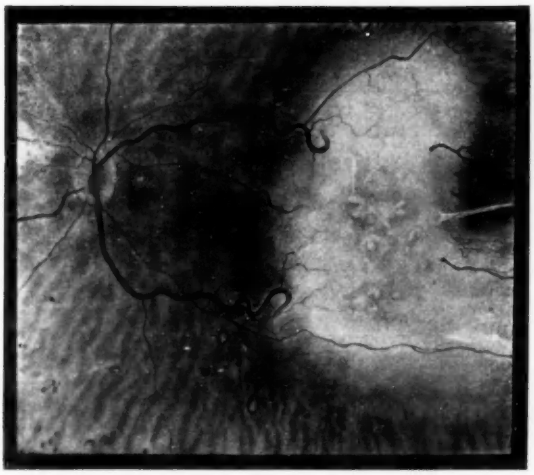

FIG. 5.—(Pfeiffer).—Photograph of colored drawing of the left fundus of the ninth case, showing the tumor with typical cell clusters.

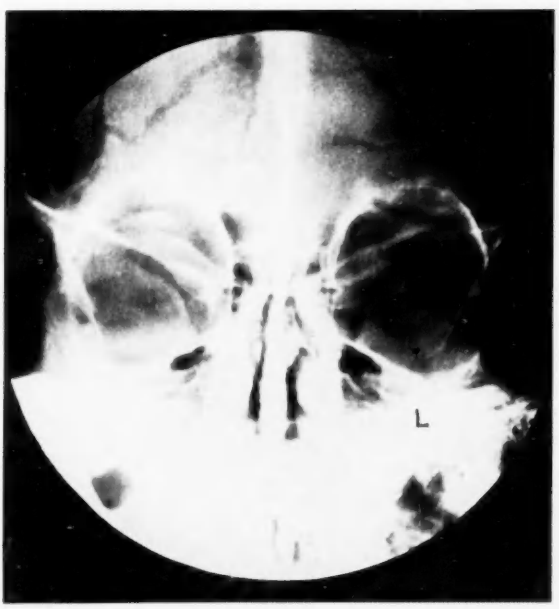

FIG. 6.—Roentgenogram of the orbits of the ninth case showing the shadow of calcium in the left.

CASE IX (456985) — *Unilateral retinoblastoma, unproved. Typical calcium deposits shown on orbital and bonefree roentgenograms. Enucleation refused by parents.* L. W., a boy of 3½ years, was observed by his mother to have "something" wrong with the left eye nearly three years before present examination. Externally both eyes were normal. The left fundus showed a well-outlined bluish white mass in the temporal half of the vitreous chamber, which was elevated approximately 10 diopters. Two large arteries disappeared in the mass and an area of the surface showed ulceration of the internal limiting membrane with typical so-called glioma-cell clusters. The surrounding fundus showed granular pigmentary degeneration, suggestive of old chorioretinitis. There was some interference with transillumination (Fig. 5). This lesion was regarded by several competent observers as inflammatory in origin, and related to Coats' disease. Roentgenograms of the orbits revealed a well-defined, mottled, and irregular shadow of calcium in the left, measuring approximately 4x6 mm., apparently within the globe (Fig. 6). To prove further the position and nature of the shadow, the child was given an anesthetic, and the eye was grasped, rotated nasally and pulled outward to place the tumor between the film and the tube, and bonefree roentgenograms made (Fig. 7). These films revealed distinctly the typical granular nature of calcium deposition in a retinoblastoma. Removal of the eye was advised, but refused by the parents.

CALCIUM IN OTHER OCULAR CONDITIONS

Calcium may be deposited in eyes in other diseases, and roentgenographic shadows of such deposits must be differentiated from shadows of the psammous bodies of retinoblastoma. Deposits in senile eyes need not be considered, for they are out of the age limit for the occurrence of retinoblastoma, and would not be confused. Heteroplastic bone in the choroid occurs usually in older patients in atrophic or phthisical eyes (Fig. 8). Roentgenographic shadows of bone in choroid have fairly regular margins and density, and are crescentic, though the shape depends on the angle of projection. Calcium in the lens is revealed on the film by the roentgen ray as a globular mass, or if the cortex only is calcified, as a small curvilinear shadow not more than twelve millimeters in diameter, and is commonly found coexistent with bone in the choroid, but may be found in an otherwise apparently uninvolved eye. Calcification of luxated lenses

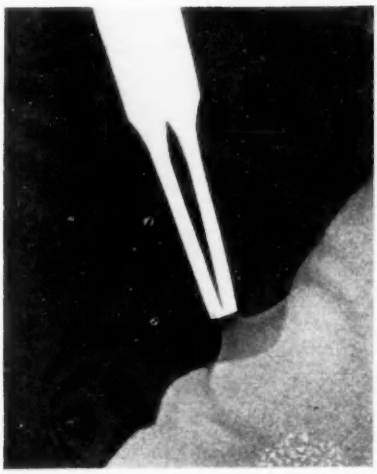

FIG. 7 — (Pfeiffer) — Drawing of bone-free roentgenogram of the left eye of the ninth case, showing the deposits of calcium in the lower right corner.

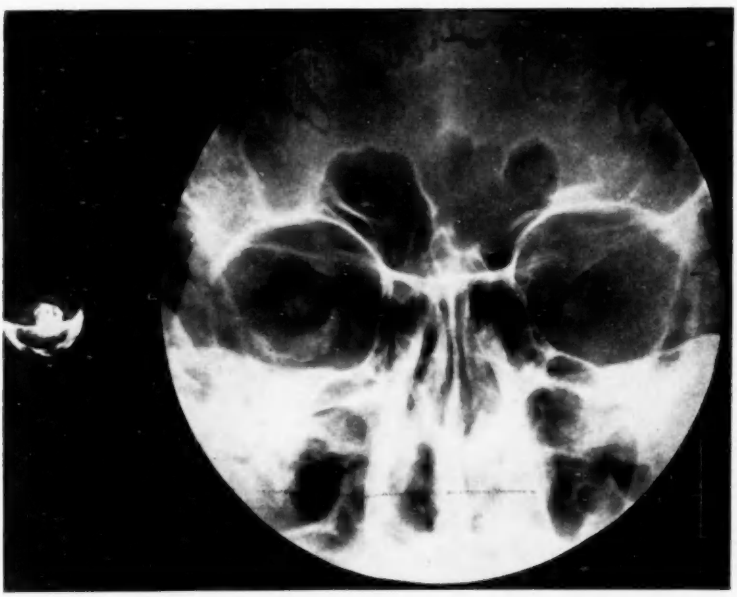

FIG. 8 — (Pfeiffer) — Roentgenogram of orbits showing typical shadows of calcium in the lens and of heteroplastic bone in the choroid. A roentgenogram of the enucleated eye is shown on the left.

is not uncommon. The shadow of calcareous degeneration in retinoblastoma is characteristically mottled and irregular in outline. An eye affected by retinoblastoma may shrink and atrophy and be regarded as a pseudo-glioma. Thus, roentgenographic study of such eyes should indicate the true nature of the condition.

TECHNIQUE

The usefulness and success of this method is directly proportional to the roentgen technique employed, for only in films of good detail can the sometimes minute shadows be detected and differentiated. Stereoscopic films are of utmost value and should be made of the orbits with the patient's head in the Caldwell position for sinus examination. The exposures should be made with the patient under general anesthetic. If instantaneous exposures ($\frac{1}{20}$ second) are used with the large focal spot of the tube, the tube-cassette distance should be increased to at least 36 inches and a small cone attached. The Pfeiffer[3] device for projecting the optic canals is used in our laboratory because it aids greatly in positioning and immobilizing the head, and at the same time reveals the optic canal on the film.

The bonefree technique for roentgenographic examination of the anterior segment of the eye advocated by Vogt[4] should be used in conjunction with the usual projections as in foreign body localization. Films of this method reveal minute bodies of slightly more density than that of the tissues of the eye. Such films are so delicate that several granules in a retinoblastoma may be revealed by them if the eye is rotated in such a fashion as to interpose the tumor between the tube and the film (Fig. 7). The retro-orbital injection of novocaine or other non-irritating fluid, or the injection of air in Tenon's capsule, may be used to proptose the eye to expose more of the globe to the ray.

SUMMARY AND CONCLUSION

Calcareous degeneration in retinoblastomata can be shown by the roentgen ray and is revealed on the film by characteristic mottled or granular shadows, which are cast by the sandy bodies in the tumors. It is shown that calcareous degeneration is frequent in retinoblastoma and varies in extent. Seventy-five per cent of 20 laboratory specimens contained calcium in some degree on the roentgenograms. The specimens were not whole globes and accurate deductions could not be made on the relationship

between the size and age of the tumor, and the amount of calcium present.

The application of this observation is well shown in the roentgenographic study of twelve patients with clinical diagnosis of probable retinoblastoma. Eight of the twelve were proved histologically to have retinoblastomata and six of these showed typical shadows of calcium in the orbits before enucleation. Four of the eight had bilateral retinoblastomata and three of these showed calcium in both orbits. Therefore, nine of twelve eyes with retinoblastoma, granting the associated and unproved eyes in the bilateral cases were retinoblastoma, showed calcium in globes in situ. The two retinoblastomata which failed to show calcium were in the first stage; one nearly filled the vitreous chamber. The four pseudo-gliomata showed no calcium in the orbits by roentgenography before or after enucleation.

The roentgenographic shadows of psammous bodies in retinoblastoma are differentiated from shadows of heteroplastic bone in the choroid and other calcium deposits in eyes.

In is contended on the basis of this work, that approximately 75 per cent of retinoblastomata undergo sufficient calcareous degeneration to be recognized roentgenographically, and that the granular and irregular shadow is pathognomonic of tumor when found in children.

* * * * *

I should like to express my appreciation for the aid and suggestion of Professor John M. Wheeler and Doctor A. B. Reese, and to the several attending surgeons of the Institute of Ophthalmology of the Presbyterian Hospital, who permitted me to study their patients.

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DISCUSSION

Dr. A. B. Reese, New York: The diagnosis of retinoblastoma cannot always be made without some reservations. Sometimes the enucleation of a sightless eye is warranted even when such a lesion is only remotely possible. For this reason, about one out of four eyes removed with the probable clinical diagnosis of a retinoblastoma prove by microscopic examination to be a pseudo-tumor. Therefore, any aid in the differential diagnosis is quite welcome.

Judging by the series of cases studied by Doctor Pfeiffer, he offers us a valuable means of establishing the diagnosis in about 75 per cent of the cases. The absence of the characteristic calcium deposits in the roentgenogram does not rule out a retinoblastoma, but their presence is more than presumptive evidence.

The only conditions that have given confusing pictures are heteroplastic bone in atrophic globes and calcification of the lens. Both of these have been easily differentiated from the diffuse, mottled, flaky appearance of calcium in a retinoblastoma.

Sometimes with the ophthalmoscope one sees confluent chalky white areas over the surface of a retinoblastoma. I believe these represent denseness produced by superficial calcium deposits and are only seen in a retinoblastoma.

Although Doctor Pfeiffer's conclusions are based on a limited number of cases, they are sufficiently encouraging to prompt others to employ his method. In due time there should be enough data at hand to warrant a final evaluation.

Dr. Mary Knight Asbury, Cincinnati: The printed abstract of Dr. Pfeiffer's excellent paper stimulated me to review my collection of retinoblastoma.

Fifteen eyes, or fifteen patients, were represented. As I have not had such an eye to examine for several years, the specimens were all old and I have no x-ray data on them.

Microscopically, eleven showed calcium deposits in the tumor, four did not. This tallies very well with Dr. Pfeiffer's figure for the occurrence of calcareous degeneration. But in my short series, the presence of calcium seems to be related to the stage of the disease. Five eyes had been enucleated in the early stages of the disease, before there had been any extension of the new-growth into the optic nerve, or other extraocular extension. In four of these five eyes no calcareous degeneration was found in the few sections examined. Complete serial sections were not available. Two of the four patients represented by these eyes were alive and well four and five years after enucleation. I have no subsequent history on the other two, nor on the one early case which showed a few calcium deposits. All the eyes with involvement of the optic nerve and with extraocular extension showed calcareous degeneration in the tumor, although in two there were only a few small deposits.

My small series would lead one to conclude that the presence or absence of calcareous degeneration in retinoblastoma as demonstrated by the x-ray is of little diagnostic assistance in the early cases in which one might hope for a favorable outcome following enucleation.

MASSIVE RETINAL FIBROSIS IN CHILDREN

ALGERNON B. REESE, M.D.*

NEW YORK

WE HAVE encountered several cases characterized by a grayish-white mass protruding from the retina in young individuals due to the organization of hemorrhage occurring at birth or from trauma after birth. The fundus picture resembles that of retinoblastoma, from which it must be differentiated. The nature of three of the cases can be proven rather conclusively, and these will be reported in detail.

REPORT OF CASES

CASE I

History.—A boy, age 6 years, had normal vision in both eyes at a routine school examination in September, 1931. A few days later he was struck by an automobile and received a laceration over the left side of the forehead, and the left eye was bandaged. No examination of the eye was made at that time. One year later, in September, 1932, at a routine school examination, poor vision was noted in the left eye.

Clinical Examination.—*Vision* of the right eye with correction was 20/40; of the left eye, 2/200. The iris of the left eye was somewhat darker than that of the right eye.

Fundus Examination of the right eye was negative. In the left eye there was seen, adjacent to the disc, temporally, an elevated yellowish to grayish-white mass about two disc diameters wide and six disc diameters long, with the long axis vertically placed. Retinal blood vessels coursed over the elevation and a few new-formed vessels, or dilated capillaries, were seen over the surface. The margin of the lesion was irregular in contour and fairly sharply demarcated by a lighter border in which were many punctate white dots. These same dots were noted elsewhere over the fundus with a tendency to accumulate in foci. They were particularly large below in the periphery. No vascular changes of note were present. The disc was red and its margins indistinct.

* From the Department of Ophthalmology, Columbia University. Cases I and II are from the services of the Eye Institute and Case III is available through the courtesy of Dr. D. H. Rhodes of Pittsburgh.

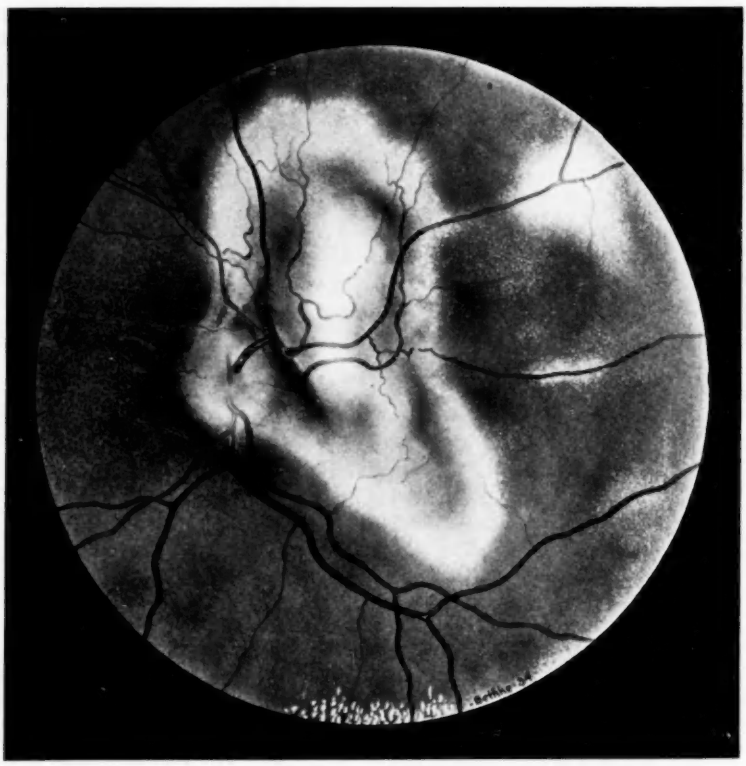

FIG. 1 — (Reese) — Fundus drawing of Case I.

The lesion was observed over a period of a year, during which time it was thought to have become more extensive, and enucleation was advised because of the possibility of retinoblastoma. The *physical examination* was entirely negative. A Wassermann reaction was negative. Calcium could not be demonstrated in the globe by roentgenogram.

Microscopic Examination — On the temporal side of the disc was a large fibrous mass which extended from the external retinal layers into the subretinal space. It was sharply demarcated and composed of dense, fibrous tissue, rather sparse in nuclei. It was surrounded by a neuroglial capsule which was particularly thick at the nasal border. Two types of pigment were noted in the lesion. One consisted of phagocyted pigment granules from the pigment epithelium and the other of amorphous crystals which gave a positive Pearl stain for hematogenous pigment. An occasional lymphocyte was noted. The overlying retina was adherent over most of the internal surface, though a flat detachment

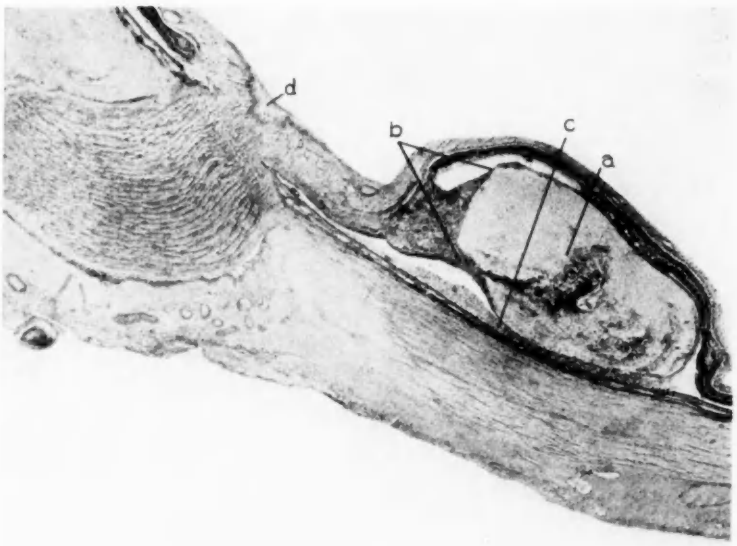

FIG. 2 — (Reese) — Microscopic section of Case I. *A*, dense fibrous mass throughout which are areas of pigment; *B*, neuroglial capsule; *C*, proliferated pigment epithelium; *D*, disc with physiologic cup bridged by fibrous membrane.

was noted in the nasal periphery. The underlying pigment epithelium had proliferated into excrescences and many free cells which formed a layer under the lesion. The retina contained cystic spaces in its various layers. Most of them were divided into subdivisions by delicate septa which seemed to be the walls of fat cells. Some were filled with foam cells resembling the embryonic type of fat cell. Others contained a faintly eosin-staining homogeneous fluid. The optic disc was somewhat edematous, and bridging over the physiologic cup was a fibrous membrane. At the nasal side of the disc there was a hyaloid excrescence of the lamina vitrea. The choroid was not materially changed in the vicinity of the retinal lesion or elsewhere. The anterior sector of the globe appeared to be normal.

Remarks — The white dots noted over the fundus with the ophthalmoscope represent cystic spaces in the retina filled with fat or an albuminous fluid. The indistinct margin of the disc was due to a fibrous membrane over its surface bridging the physiologic cup.

CASE II

History — A boy, age 5 years, had been blind in the left eye, so far as the parents knew, since birth. Forceps were used to aid delivery. Inflammation had never been noted in either eye.

Clinical Examination — Vision in the right eye was 20/30; in the left eye, hand movements. He could not fix with left eye which diverged 35 degrees. The *fundus* of the right eye was negative. In the left eye, just temporal to the disc, was an elevated grayish to yellowish-white mass with the long axis extending temporally in the horizontal meridian. The mass branched upward and downward forming roughly the letter T. Retinal vessels coursed over its surface and around the base were either some new-formed vessels or dilated capillaries. Some of the ves-

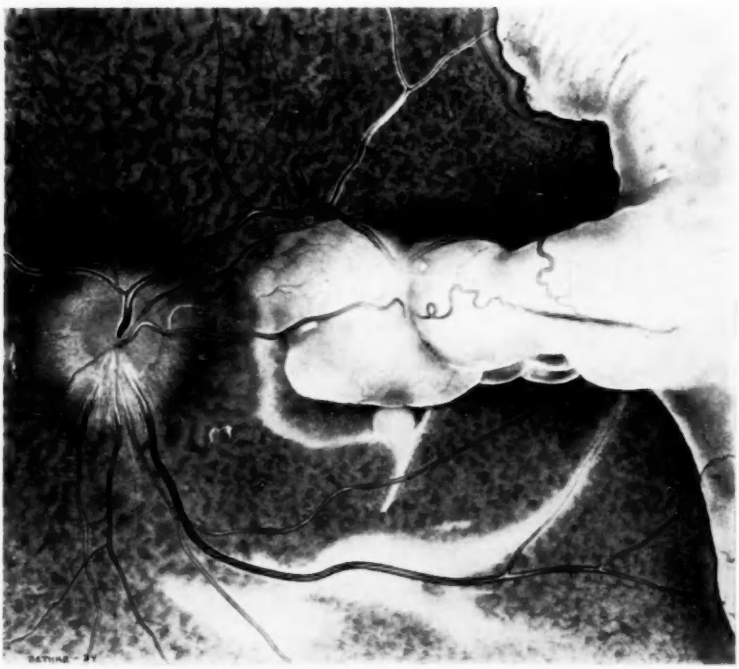

FIG. 3 — (Reese) — Fundus drawing of Case II.

sels in vicinity of the mass showed perivascular white streaks. Below the mass was an irregular white, flat area apparently lying under the retina. After four months the lesion had apparently progressed and enucleation was advised because of the possibility of retinoblastoma.

The *physical examination* was entirely negative. A Wassermann reaction and Schick-Mantoux test were negative. Radiographic examination of the skull was negative. Calcium could not be demonstrated in the globe by roentgenogram.

Microscopic Examination — From near the temporal border of the disc there appeared in and under the retina, large irregular masses of fibrous tissue of two distinct types which were rather sharply demarcated one from the other. The one was lighter-staining, more homogeneous, dense, and rather sparse with nuclei. This seemed to be in close relation to the pigment epithelium and was probably laid down as a result of its proliferation. In it were large plaques of readily recognizable pigment epithelium growing in cords which coursed mostly parallel, and in places the inter-

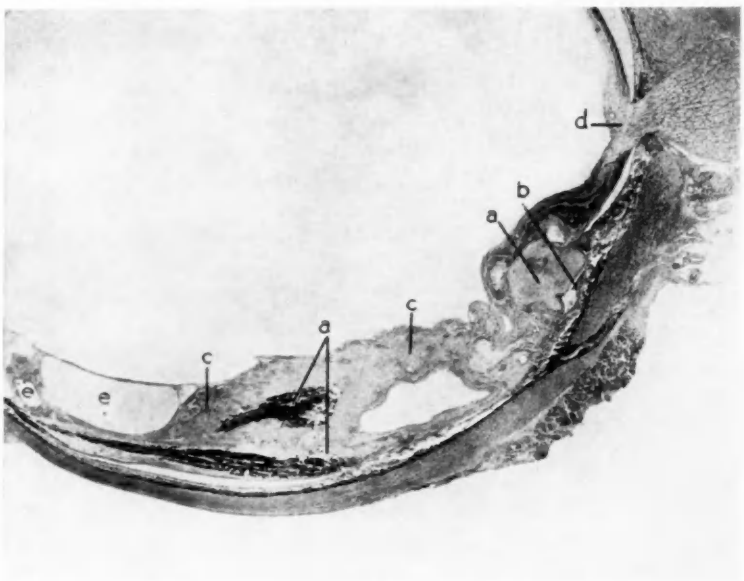

FIG. 4 — (Reese) Microscopic section of Case II. *A*, dense homogeneous fibrous tissue laid down by pigment epithelial proliferation; *B*, bone; *C*, neuroglial proliferation; *D*, disc; *E*, contracting bands forming cysts.

nuclear element was hyaloid in appearance. There were some areas of bony degeneration present. The other type of fibrous tissue stained more intensely with eosin, was much richer in nuclei and more fibrillar. This tissue was adherent to, and more or less enveloped the first mentioned type, and represented a neuroglial proliferation of the retinal supporting tissue. Only remnants of the nuclear layers were seen. Throughout both types of fibrous tissue there were cholesterin crystals and small remains of old blood. The two types of pigment were seen diffusely scattered or in foci. The one was phagocyted pigment from the pigment epithelium, and the other hematogenous pigment which gave a positive Pearl stain. A few scattered lymphocytes were noted.

Contracting bands, particularly in the periphery temporally, had pulled the fibrous tissue into cystic spaces. There was no retinal detachment. The blood vessels in the lesion, and to a less extent in the retina, showed some sclerosis. The latter vessels show a very definite perivascular infiltration of lymphocytes. The choroid and anterior sector of the globe were not remarkable. The white streaks seen ophthalmoscopically along the blood vesesls represented a perivascular infiltration of lymphocytes.

FIG. 5 — (Reese) — Microscopic section of Case III. *A*, fibrous plaque. The retina is adherent to it and thrown into folds by its contracture; *B*, proliferated pigment epithelium; *C*, cysts of retina; *D*, artefactous detachment of internal limiting layer of retina; *E*, artefactous detachment of retina from choroid; *F*, optic nerve cut tangentially.

History — A boy, age 7, had not been able to count fingers or recognize objects with the left eye for years. This eye had converged for some time. There had been no pain nor redness in either eye. The child was delivered with difficulty by aid of forceps. He was adopted at the age of nine weeks, the foster mother stated that there was a forceps mark across the left brow, pink in color, gradually changing to brown, but the mark had disappeared in the course of a year. There was also a drooping of the left upper lid which disappeared in about two months. The mother's Wassermann reaction was negative.

Clinical Examination — The *vision* of the right eye was 20/20; that of the left eye was reduced to perception of hand movements. There was left esotropia of 45 degrees. *Fundus exam-*

ination of the left eye was negative. In the left eye there was a grayish-white elevated mass surrounding the disc which extended in fan shape nasally and temporally. On the temporal side it extended beyond the macular region and the disc so that only about one-third of the disc circumference was visible. Many new-formed blood vessels were seen on the surface of the mass.

Microscopic Examination—Adjacent to the disc, and extending more temporally than nasally, was a fibrous plaque which appeared to come from the external retinal layers and extended out under the retina. It was adherent to the adjacent choroid because of pigment epithelial proliferation which extends into it. Throughout the fibrous tissue there was some free and phagocyted epithelial and hematogenous pigment. A few cholesterin crystals were noted. There was no inflammatory reaction except an occasional lymphocyte. The surrounding retina was drawn into the contracting fibrous lesion. There were some quite large and small cystic spaces in the retina temporal to the lesion. These were located primarily in the external plexiform layer and were filled with eosin-staining granules. There was an artefactous detachment of the retina from the choroid and of the internal limiting layer of the retina. The retinal blood vessels showed some perivascular infiltration of lymphocytes.

* * *

The organization of retinal hemorrhage produced at birth, or by trauma after birth, seems to be the etiological factor in these cases. In cases I and III, there is certainly the history of sufficient trauma to the involved eye. In case II, there is only the history of instrumental delivery. Retinal hemorrhages, though, in the new-born with normal delivery are not uncommon because of the congestion produced in the jugular area, the veins of which have no valves, thus allowing the full force of the pressure of the congested vessels to be transmitted to the weaker terminal vessels. Sharpe and Maclaire have found bloody spinal fluid in forty-six out of five hundred consecutive new-borns. They conclude, therefore, that intracranial hemorrhage occurs in approximately ten per cent of the new-borns, and in most instances it is unattended by any sign which would cause it to be suspected. Factors which seem to predispose to intracranial hemorrhage are prolonged labor, especially when forceps are used as a last resort instead of early, premature deliveries, breech extraction, spontaneous or precipitate births, too strenuous efforts at resuscitation, asphyxia, interference with fetal or placental circulation, such as

pressure upon the umbilical cord and premature separation of the placenta.

It in interesting to note that the ten per cent of intracranial hemorrhages of the new-born reported by Sharpe and Maclaire corresponds to the ten per cent retinal hemorrhages of the new-born reported by De Vries and others. Some of the intracranial hemorrhages of the new-born are quite massive and lead to death or permanent serious involvement of the function of the central nervous system, particularly cerebral spastic paralysis associated with mental retardation. As the retinal circulation is a continuation of the intracranial system, it is not unlikely that similar massive hemorrhages occur occasionally in the retina of new-borns, and that organization leads to the type of condition described in this paper.

The evidence that the lesion in these cases has resulted from the organization of hemorrhage seems to be sufficient. The Pearl stain showed the presence of hematogenous pigment, or hemosiderin, in cases I and II. No such stain could be employed in case III because of the lack of sections.* The lack of inflammatory reaction in and around the retinal lesion, and the presence of cholesterin in cases II and III, also supports the thesis of organized hemorrhage as the basis for the lesion.

The primary site of the hemorrhage in these cases seems to be the external plexiform layer of the retina which is the layer in which retinal vessels terminate. From here the hemorrhage may spread out in the external layers of the retina and particularly to the subretinal space. The adjacent pigment epithelium proliferates into fibrous-like plaques uniting the retinal lesion to the choroid. This accounts for the fact that in no one of the cases was there a retinal detachment. It is quite possible that an extensive lesion in the later stages when more contracture takes place, could produce a detachment of some of the adjacent retina, or the retina together with the choroid. Also, the gradual contracture of the fibrous tissue can pull into it more and more of the adjacent retina and thereby give the lesion the appearance of progression. This occurred in cases I and II, and led all the more to the suspicion of a retinoblastoma. The lesions were all in the vicinity of the macular region which apparent predilection

* This stain would give a similar reaction for siderin occurring as the result of a foreign body, but there is no evidence that a foreign substance entered the eyes.

cannot be accounted for unless it is because hemorrhage is loathe to absorb in this relatively avascular area and is therefore organized.

In adults, trauma seems more likely to produce hemorrhage not at the terminal vessels in the external layers of the retina, but from the larger vessels in the internal layers. The hemorrhage, therefore, tends to spread not under the retina but inward toward the vitreous. In such instances, if the hemorrhage remains within the confines of the internal limiting layer of the retina, it probably is more readily absorbed than in the avascular external layers or subretinal space. Any organization of it leads to detachment of the retina for there is no choroidal reaction to hold the retina and prevent its detachment. If the hemorrhage gets beyond the confines of the internal limiting layer, it enters the vitreous and either absorbs or is organized, giving the picture of retinitis proliferans.

There are other conditions due to retinal hemorrhages which organize into fibrous masses similar in some ways both clinically and pathologically to the type described in this paper. Among these can be mentioned exudative retinitis of Coats, retinitis circinata, and disciform macular degeneration of Kuhnt-Junius. Metastatic ophthalmia can simulate the condition ophthalmoscopically.

Coats ascribed all of his cases to the organization of hemorrhage in the external layers of the retina and the subretinal space. The underlying causes of the hemorrhage varied in the different cases. In some he thought it due to an abnormal character of the blood, in others to vascular defects which may have been congenital, while others must have occurred at birth or from trauma.

De Wecker and Ammann have shown that the underlying factor in retinitis circinata is hemorrhage in the external layers of the retina. There are a number of reports in the literature of cases in which typical retinitis circinata occurred in one eye and a large white retinal mass in the other eye in which no retinitis circinata was present. Fisher has called attention to the similarity of these cases clinically and pathologically to those described by Coats and the same similarity exists to the cases described in this paper.

CONCLUSION

Retinal hemorrhage in children can organize into a fibrous mass resembling clinically a retinoblastoma. Due to contracture, more and more of the retina can be pulled into the lesion giving it the appearance of progression, and thereby leading all the more to the suspicion of a retinoblastoma. There is usually no detachment of the retina because the lesion is adherent to the choroid. Retinal hemorrhage occurs at birth from the same conditions that produce the analogous condition of massive intracranial hemorrhage. It may occur as the result of trauma in early childhood.

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DISCUSSION

Dr. Mary Knight Asbury, Cincinnati: When Dr. Reese sent me the material he has just presented, I was struck by the beautiful correspondence between the fundus drawings and the photomicrographs. Anyone who studies these pictures carefully will certainly view the next similar case with more intelligent interest.

The clinical picture usually spoken of as pseudoglioma is not always produced by the same etiologic factor. Last winter several cases of metastatic ophthalmia following meningitis were seen in the eye clinic. The milder ones with little or no involvement of the anterior segment fitted well into the category of pseudoglioma. So far, none of these eyes have been enucleated, so the microscopic findings are not available for comparison.

In attempting classification, the pathological picture has been renamed so often that there is great confusion of terms. Frequently eyes examined microscopically and diagnosed exudative retinitis (Coats' disease) are accompanied by inadequate histories, making it impossible to reconstruct the story and designate the causative agent.

In this instance, the three cases are analogous clinically and microscopically, and the choice of the term "massive retinal fibrosis" is a happy one, vividly and accurately descriptive. I agree with the author's conclusion that the histories and the microscopic pictures point to trauma as the exciting cause and retinal hemorrhage as the basis of the pathologic changes.

DR. E. M. JOSEPHSON, New York: Dr. Reese's time was somewhat limited. I rise to ask if he will be so kind as to spend a little time on the differential diagnosis.

I was particularly interested in his mentioning in the abstract the group of conditions described by Coats. As I recall it, most of these cases described by Coats, in his article, showed the inflammatory reaction. Most of them showed involvement primarily of the blood vessels. You will note from the pictures Dr. Reese showed here, the blood vessels overlying the mass in the retina were pretty much intact. One picture showed some slight variation in the lumen of the vessels.

As you know, the group of cases described by Coats have since then been differentiated between a number of diseases. If my memory serves me right, the condition Dr. Reese presents distinguished itself sharply from the group of cases described by Dr. Coats in that there was no inflammatory reaction and the vessels are not primarily involved.

DR. ALGERNON B. REESE, closing: In regard to Dr. Josephson's comments about Coats' disease, Coats ascribed all of his cases to the organization of hemorrhage in the external layers of the retina and the subretinal space. The underlying causes of the hemorrhage varied in the different cases. In some he thought it due to an abnormal character of the blood, in others to vascular defects which may have been congenital, while others must have occurred at birth or from trauma.

I believe in going over these cases he also evidently included some cases of metastatic ophthalmia. In other words, the term "Coats' disease" does not represent a disease inherency in the strict sense of the word, but it represents a heterogeneous group of cases.

OPHTHALMIC ERRORS

HANS BARKAN, M.D.

SAN FRANCISCO

A DEAL OF temerity is needed to approach this subject: we dislike to think of our errors, though occasionally viewing those of our confreres with a certain amount of sympathetic satisfaction: I can only say in excuse of the title that the essayist has committed all the allowable errors and some of the inexcusable ones at various stages of his career, and that with this admission may he be pardoned if he points out the mote in the eye of his confreres, admitting the beam in his own, willingly?

A certain number of us have made our first error in ophthalmology by becoming ophthalmologists in the first place. We were led into the field by practical expediency or fortuitous circumstance and have done our best. But if ever a field of medicine required special aptitude, it is that of ophthalmology. Without a gentle and unusually dexterous hand, and, if possible, bimanual dexterity, many errors, and, with the best of will, repetitions of the same error occur. Without a natural disinclination to interfere drastically in any happening, event, or course of proceeding, there may be too much meddling with too many drugs or instruments; if of too sanguine a temperament, too much reliance on the spontaneous cure of cases, aided perhaps by one particular fetish of a medication. One may fail to give enough meticulous examination, or perhaps ignore microscopic evidence warning us of trouble to come. One may be of too pessimistic a turn of mind, not inclined to let natural healing processes alone, unduly frightened of an occasional extra tear or injected vessel, adopting a new medication for every trivial change in the clinical picture. Finally, the eye may get well in spite of you, or refuse to heal because of constant well-intentioned but apprehensive meddling. If we lack the quality of making fine distinctions mentally between right and wrong, between moral and immoral, between music and noise, between what is genuine in art or literature and what is hokus pokus, — if, in other words, having the common good sensible practical head of the average highly competent individual, we are going to make many errors in our field — we then do not

possess that sensitivity of intellect needed, I believe, to appreciate properly the innumerable shadings of ocular disease. When we realize that this finesse of judgment and action based on it has to be built up on a wide basis of general medical knowledge, we pause before the really great and nearly errorless members of our profession, past and present, in humble admiration of their qualities as men and as scientists.

Errors due to insufficient training are perhaps the hardest to get rid of, due largely to the fact that we do not know that we possess them. They are almost positive liabilities instead of negative factors. They occur again and again, followed always by the same bad results, and unfortunately what Jack has not learned as a boy, John will not acquire as a man. A violinist who has not learned the proper and convenient finger positions as a boy, as a man leaps about the strings in frenzied attempts to get notes into a certain space of time. The man who has not acquired as an operator what the Germans call "Schule," is distinguished mainly by his lack of proper tempo. The operation is hurried in one detail, slowed up for another — each step looks as if it were the operation and not to be followed by another logical move — all is disjointed. It is a good deal like the pulling of a tooth in irregular hurried and spasmodic jerks. The door for grievous error is wide open. As a refractionist a lack of physiological knowledge makes him the slave of his complicated tools. His machinery dictates the prescription, instead of being an accessory to his appreciation of his patient's particular needs, taking into account his patient's age, mentality, working hours and distance, general physical well-being, past history in regard to glasses and future plans in life.

The commonest error of many of us is over-correction of the presbyope. We move him up too close in response to his demand for better near vision of small print, thereby increasing the effort at convergence, and fail to realize that as accommodation vanishes so the reciprocal innervation between convergence and accommodation is disturbed and convergence is now physiologically unwilling to be called upon so drastically.

Frequently we are distressed by the fact that our prescription as written is not as the glasses prove to be and one may have committed an error. Often it is a result of the "hurry" of our office, of not having centered the trial frame accurately, and of not having measured and recorded the distance between the posterior surface of our trial lens and the corneal vertex. In low

degrees of correction it is of little consequence, but in any correction approaching or exceeding 5 D., it is of decided importance. Another common error is not to ascertain the P.P. and P.R. of each eye separately. Occasionally, there is a marked discrepancy which not only has a bearing on the prescription but indicates a formerly unsuspected neurological condition. The myope, usually free of subjective complaints, except that of poor vision, must not be led into trouble. Never correct a high myopia, not formerly corrected, too fully. Sensitive people feel it as a psychological insult to have the radius of their formerly rather restricted visual circumference suddenly increased tenfold, with mental reactions so far unused suddenly called into constant visual activity. We can often obtain comfort and the necessary visual acuity when needed by giving ½ to ⅔ of the distance correction, and then add a monocular lens of — 5 D. or — 10 D. to be used as an accessory lens. Professor Löhlein of Freiburg recently mentioned the case of a general practitioner, with a myopia of 34 D., who could comfortably wear only — 20 D. and had difficulty in finding the houses of his patients. With a monocle of — 10 D. added on these occasions he could find the house number and could continue in practice comfortably.

In cases of high hyperopia or aphakia with amblyopia, better vision can occasionally be obtained by holding a + 10 D. lens about 10 cm. in front of the corrected eye, thereby through enlargement of the retinal image obtaining as high as twice the distance vision obtainable by the cataract glass alone. By uniocular reading, so as not to exert convergence, addition of appropriately strong convex glass bringing print as close even as 10 cm. may enable patients to read J 1.00, whereas with their ordinary reading correction they could read only J 4.00. In some cases this enabled them to continue their life work. When combined with a music stand on which the book can be placed, so that it must not be held close or the head lowered, addition of strong convex lenses with an appropriate prism base in may afford comfortable reading when all other means have failed.

It is an error to suppose that all headaches are due either to the right eye, the left eye, or both eyes. Often the patient's money and our time is saved by speaking to the family physician after the first visit. The history is often illuminating in this regard: headaches occur, say, only with the periods, or only in the morning and are relieved by a cup of coffee and moving about; or only when stooping, or after a cold, or only after worry or

psychical excitement. I have often prevented the error of false valuation of symptoms by saying to patients inclined to many complaints, "Suppose I were a magician and simply by waving my hand could completely rid you of *one* complaint, which would you choose to be rid of?" It works well frequently.

In pointing out what seems to me a frequent error in therapy, —though here, naturally, we are in the most empiric of fields, with so much play of prejudice, likes and dislikes, conservatism, radicalism or liberalism, and with so little to cling to scientifically that the errors fairly bristle — may I speak of tuberculin and the errors to which it leads? I do not refer to its use primarily nor to the question as to whether the process it is given for is tuberculosis in the first place, but the giving of it for long periods when the process is not improving, nay, in many instances, vision becoming poorer. I have repeatedly seen the use of tuberculin continued when certainly had we been dealing with another drug or another disease, we would have acted differently. Only recently a patient for whom iodides were prescribed and a daily slight elevation of temperature by putting his arms into very hot water for fifteen minutes improved remarkably for the first time in a year. Here, too, may be mentioned the error of looking forever for the etiology of, say, an exudative, sluggish, recurring iritis; out go all focal infections; the Wassermann is negative; tuberculin is used and the patient appears at innumerable conferences and demonstrations. Meanwhile, it has not occurred to anyone to shorten the process, in many cases even to stop it, by an iridectomy done in a quiescent interval. Some of my most satisfactory results have been in patients of this type, though to this day I do not know what the etiology was.

A very small selection of drugs will go a long way in ophthalmology. Hopping frantically from one to many will not get us very far. It is better to concentrate on a small number of standard drugs. To illustrate with pilocarpine: I have seen a two per cent solution hold tension below normal but be painful and interfere with vision; a ¼ per cent solution served in this patient just as well and with lack of complaints, just as occasionally a regulation of tension can be secured with a 6 per cent to 8 per cent solution of pilocarpine when weaker solutions have failed.

A word regarding dionin. Some patients sneeze after its use. I was shown a patient who in so doing immediately noted poor vision, the result of fresh retinal hemorrhages. I have seen

the same result after diathermy. Be careful, then, to instruct people with arteriosclerotic vessels and hypertension to press on the tear duct for some minutes after the use of dionin. Ringer's solution has proved useful to me in a variety of corneal dystrophies. These patients often complain of "dry" eyes. One patient of mine could not and had not wept for years. She was seen in consultation by Professor Lindner, who sent her home to smell onions! This not working, to eat horseradish! This not working, to drop Ringer's solution in her eyes very frequently, with satisfactory results. This leads me to believe that we are committing an error in not buffering solutions to the p_H of the tears, in many cases, and while I have not yet done so, I will try it. Personally, I consider it a waste of time, therefore an error, to use vaccine therapy, at least in cases of staphylococcus blepharitis and the recurring forms of hordeolum. Success is certain here if we inject novocaine along the hair roots, pull out all infected ones at one session, dip a tooth pick in 1 per cent silver nitrate and insert it in the infected opening and then follow with two to three applications of water-cooled quartz lamp.

To consider every chronic, slight but annoying, conjunctivitis a sequence of bacterial infection is an error. A number are undoubtedly of allergic origin and the more treated, the worse they become. Do not forget the value of amyl-nitrite, used cautiously in older people, not only in severe cases of ophthalmic migraine, but in atrophic stages of chorioretinitis, in pigmentary degeneration of the retina and in arteriosclerotic atrophy of the optic nerve. Cordes and Horner have recently shown the value in toxic amblyopia of the use of vessel-dilating drugs such as sodium nitrite. Finally, as regards therapeusis; please remember that we are playing a duet with our patient. We may be first fiddle, but if we are drowning out our second fiddle, let us ease up on our treatment and give him time to recover from our too energetic tone; sometimes a pause for a few days helps the second fiddle enormously.

The field in which errors are most to be condoned, it seems to me, is that of glaucoma; excused to ourselves at any rate because, after all, we can say, "As long as I do not know the reason for the fact that this eye is harder than normal I can only guess, with a certain percentage in my favor, what is wisest to do." Provided we employ common sense in estimation of age of the patient and expectancy of life, of whether one eye or two eyes in considering type of operation, of whether the disease is

chronic with relatively low tension, or acute with rather high tension, we have only one comparatively new method by which to avoid a good many of our former errors. I refer here to the curve of tension as ascertained by five or six tension measurements a day for several days, preferably with hospitalization so as to obtain an even psychological background. This curve will show us the period, usually the morning, of the highest tension and the period of the lowest when measured on the untreated eye and will furnish us with the proper basis for the use of miotics and at what time they are to be administered. The curve is most useful when compared with the curve of the unaffected eye, if such there be. It also brings out one most important feature, and that is the normal tension for the particular individual, at least in cases in which a comparison between a normal eye and an affected eye is possible. After successful operation, the taking of these curves every six months is to be advised. We see eyes slowly lose vision in a year or two following an operation, which at the time reduced pressure successfully. An occasional taking of the tension months or years afterward will not disclose a period of the day when the tension may be nearly as high for a few hours as it was before operation.

Let us not fall into the error in referred cases from other specialists of accepting their fields and their report of the tension as being representative of what that patient's tension and fields may be when we see them. I recently saw a patient well under pilocarpine who had traveled twenty-four hours previous to seeing me and carried with him a letter from his very competent eye specialist. This letter stated that the tension had never been more than 30 McLean. I took the tension and found it 90. When the patient had recovered from the fatigue of the trip and the psychological excitement of seeing a new physician, it was 30. The difference in the measurements, however, showed me that this difference could be accounted for on a vasomotor basis and that the continuation of pilocarpine and a quiet life was preferable to an operation — especially as this patient's expectancy of life was not long. It is an error not to operate on the worse eye, even though it be blind and painless. Valuable lessons can be occasionally learned in regard to the patient and the method of later operating the one useful eye. Having said that errors in glaucoma are the most excusable among those we make, we need not pursue the subject further.

Cataract operations are a different story for the younger members of the profession today than they were for those of my age, when we started practice. Today sudden emergencies, with cause for quick judgment, and where an error in judgment means loss of the eye, do not occur frequently. How well I remember uncontrollable sudden blepharospasm, ocular muscle spasm, and sudden looking up, with gaping wound and the operative field under the upper lid. Here the lightning quick operator and the man of many operations was in demand. The change is due to akinesis, retrobulbar injection, and superior rectus suture. It is an error, and a grievous one, for anyone to operate today without using these measures with confidence. If you have not learned them, do so. If you won't use them, send your cataract patients to someone who will. It is an error to try every case as an intracapsular extraction. It is an error not to try an intracapsular extraction in the favorable group. It is an error, if doing an intracapsular extraction to combine it with a total iridectomy. It is an error to excise or cauterize every iris prolapse; some can be replaced, others left alone. It is an error to believe that every focal infection in old people has to be removed before operation for cataract. It is an error to believe that perfectly normal light projection means a roughly normal fundus. I recently operated on an eye of normal tension with splendid projection and with the only suspicion of abnormality the fact that, according to the patient's statements, the cataract had matured in three months so rapidly that he was reduced from coarse reading vision to blindness. I found a total detachment of the retina, not due to the operation, as Dr. John Weeks, acting as consultant, had noted a beginning detachment over two years ago. For many perfectly good reasons the man had never been told and did not tell me that Dr. Weeks had any records of his case.

What of specific errors not uncommonly made, not in spite of highly technical equipment but because of it? A common example is the use of the high-power objective of the slit-lamp before a general survey has been made by means of good focal illumination and ordinary magnifying equipment, say a Beebe loupe. Once I missed a tiny perforation of the iris as evidence of an intraocular foreign body. One has a highly magnified but small field and can miss seeing a portion of it altogether.

In strabismus, operating it is an error not to use the O'Connor hitch method for the cases you elect to do by shortening a

muscle. Other operations are good, this is the best. In alternating cases have the patient fix with each eye in alternation and close both eyes after fixation. Repeat this several times. If on opening both eyes, ones eye fixes after it has received the last visual impression, and the other one does so not at all or less frequently, operate first on the eye that has maintained fixation poorly. In a large number, if not of too great a degree of strabismus, this is frequently the only operation required.

Finally, a few observations without any particular logical relation to each other. Do not publish cases of interest too early. You may find within a year after such publication that the case was not what you thought and did not in any way turn out as you described it. Be chary in giving a hopeless prognosis, not only from a psychological aspect, but because not infrequently your patient to his joy and your mortification is seen a few years later seeing better than for a long time. Do not in fundus examinations, no matter how diseased the fundus, feel that there is no use wasting time in seeing what glasses will do. I have had my competent associate give a patient visual acuity of 10/10 in spite of the fact that from a look at his fundus I would have sneered at the thought that a glass would be of any benefit. Do not make a prognosis to yourself as regards vision on only one method of examination. It is astonishing how different, for instance, a cataract will look with the ophthalmoscope as compared with the slit-lamp. In one case you see no good reason for much depreciation in vision, in the other case, expect very low vision, and the vision may lie somewhere between these two extremes. Do not judge the future regarding vision by the atrophy of the optic nerve, especially in children. The prognosis looks poor and yet you see them often as adults with 20/30 vision. Do not be too hopeless about restoration of useful vision at some time just because the fundus is acutely and severely affected. It is amazing what an amount of restoration to normal anatomy with normal vision can occur in marked choked disc and in marked fresh retinitis from practically any cause. Do not, because of typical star-shaped exudate in the macula, speak of nephritic retinitis. I have seen this star-shaped exudate in brain tumor. Similar cases are mentioned in ophthalmic literature. While it is wise and proper to be sceptical, at least, regarding the cure by Christian Science of what is clinically undoubtedly melano-sarcoma of the choroid, do not forget that there are definite cases on record, substantiated by excellent men, of the self-healing of

cases of sympathetic ophthalmia, tuberculosis, cataract, and so forth, and that it is never wise to say this or that cannot occur.

The loss of patients due to errors on our part can in many cases be avoided by analyzing our training and disposition, by regulating the amount of work we can do carefully, by refusing to be hurried, and by always considering ourselves as students of our subject and not finished masters of it. Some loss in practice, of course, is beyond our control. I recently lost a patient who went to one of my confreres for a cataract operation and who told him that she wanted him to operate on her cataract because a friend of hers had had her eye successfully removed by him. It is a human and humorous reasoning and much as we may learn to control ourselves, we cannot control our patients' cerebrating processes.

The value of this paper has undoubtedly been greater for me than for you. As I wrote it, the number of errors I commit rose up before me like a well-marshalled army, too many to mention in this paper. But it has made me conscious of my faults and that is helpful. If, in reading this paper when it is published, it brings all of your errors before you, it perhaps has served its purpose.

DISCUSSION

Dr. M. H. Post, St. Louis: The consideration and analysis of errors is frequently productive of equal or greater information than the exposition of achievements. It requires courage on the part of any individual or society to frankly and openly discuss their shortcomings. Yet much can be learned from such considerations. The eye lends itself well to study. Most of its structures are open to direct examination. The field has been well cultivated. The literature on the subject is rich. Although many serious problems still remain unsolved, the fact is that errors in diagnosis should be few.

We may well divide such errors into two classifications; first, those that may be avoided by careful study of the lesion, applying well known methods of diagnosis and taking into consideration all thoroughly recognized symptom complexes; and, secondly, those of such rarity as to come seldom within the experience of the practicing physician. As an example of the first group, one might emphasize the differential diagnosis of acute glaucoma from acute iritis often with secondary glaucoma; sympathetic ophthalmia and malignant uveitis; arteriosclerotic retinitis from papilloedema with hemorrhages and exudates, and blocking of the central retinal vein or one of its major tributaries; trachoma and follicular conjunctivitis; sclerosing keratitis and traumatic leucoma of the cornea; differentiation

of the various lesions of the extraocular muscles, etc. Into the second classification might fall such relatively rare conditions as pemphigus; lattice-like keratitis; tularemia; incipient choroidal malignancy, or the various tumors of the optic nerve, to mention only a few.

The author of this paper has made timely reference to the influence which temperament plays in the diagnosis as made by the individual examiner. The optimistic physician will almost invariably lean toward the more favorable prognosis and view all symptoms in the light of such optimism, while the pessimist will constantly be struck by the serious aspect of the situation. For example, it is well known to all of us how fallible is our judgment with regard to the disabilities of those who are near and dear to us. An impersonal attitude should always be striven after. Yet in the effort to avoid such prejudices, care must be exercised that we do not swing to the other extreme.

In no phase of ophthalmic diagnosis does the temperament of the examiner play a greater part than in the investigation and correction of refractive errors. Haste, carelessness, irritability and lack of patience are responsible for a great percentage of such faulty diagnoses and should fall within the first classification. Other failures, such as those due to aniseikonia, illustrate a transitional stage. A few years ago no one could have been held responsible for failure to diagnose this interesting and unfortunate difficulty, but now, due to the beautiful investigation being carried out by Professor Ames and his confrères, its recognition will soon be considered as a matter of routine.

This extremely interesting paper has brought up many interesting points. There are three of these of which I would like to speak in particular.

One is the fitness for the practice of ophthalmology. We have a serious problem here. How do we know before we begin whether we are fitted or not? Many a man, in all probability, finds himself unfitted when it is too late to turn back. I believe that this is a point which must fall on the shoulders of our teachers. They must recognize the fitness or unfitness of candidates for the study of ophthalmology.

I hesitate to mention the second subject because it is clouded in so much uncertainty, and yet I do want to speak on one point; that is tuberculosis. I have been watching a group of about sixty such cases for the past two years, and I agree heartily with the author of the paper that we must not rely entirely upon tuberculin treatment or therapeusis in many of these cases. They yield well to surgery in quiescent periods. Excellent results are obtained in cataract extraction and irridectomy in cases which we believe, by all available methods, show positive evidence of tuberculosis.

The third factor which I might point out, which the author has spoken of, is over-treatment and the use of overly-strong medication. I believe much harm is done by that in many instances. Milder therapeusis will accomplish our object better than the more strenuous.

Errors of diagnosis in ophthalmology grow constantly less and less frequent as the various problems are solved, and proportionately the obligation upon the ophthalmologist becomes greater and greater.

Dr. Oscar Wilkinson, Washington, D. C.: My Scotch-Irish disposition usually prevents me from agreeing with anyone entirely, but I am going to concede that point this time and say that I agree with the essayist in all of his remarks. I also agree entirely with the discussor when he said that the fitness of the candidate for ophthalmology should be well analyzed by the teachers or those in charge. I think a great many men go into ophthalmology not realizing the importance and the delicateness of it, and the teachers can point out those they see are unfit and steer them into less strenuous fields of medicine with much advantage.

The early errors that we men made are largely now being eliminated. In the first place, I belong to the old school in which we had no place to be trained properly. We didn't realize the importance of case history and the advantage that the proper case history will give us in making a clear diagnosis.

There are one or two points with reference to refraction I would like to mention. As an early student of Gresham College in London, I wanted to get into something else besides refraction. The instructor insisted that I learn refraction. He said, "At least 70 per cent of your work is going to depend upon refraction and you had better learn it."

I believe today that a great many of our busy men feel they haven't time to do refractive work properly. If one considers particularly the history of his case, the nature of the work that the patient is going to do, and the condition of the light in which he is working, it will certainly aid us materially in the future comfort of that patient.

With reference to the use of strong cylinders on elderly subjects, I think we often make a mistake in trying to correct too much astigmatism in elderly subjects who have not previously worn astigmatic correctives. You are certainly going to get into trouble if you try to correct hard degrees of astigmatism in elderly subjects who haven't worn them previously.

There is one thing I would like to mention in connection with the myopics. In those cases in which the visual acuity for one reason or another is markedly reduced, if you will simply explain to the patient that he can obtain a much better vision by raising his work closer to his face than you can give him with stronger corrections, he will be very grateful to you in the long run.

Dr. Hans Barkan, closing: I wish to thank Dr. Post very much for his discussion. The paper is naturally a bit difficult to discuss because so much is left out in the paper. We couldn't possibly take in a whole paper of ophthalmic errors in a day or a year.

I was very glad to hear Dr. Wilkinson say what he did about high cylinders and very strong glasses in general. Of course, it is an old story, but I think sometimes we have to keep before our minds the fact that most patients do not come to us because they want to see better, except the milder cases. In the other group occasionally a man will say, "Doctor, I don't see well enough in the distance," a man not a myopic case, but it is rather rare. They come because they have headaches or dizziness or inability to read long.

MANAGEMENT OF COMPLICATIONS IN THE OPERATION FOR SENILE CATARACT

HARRY W. WOODRUFF, M.D.

JOLIET, ILL.

THE PROGRESS which has been made in recent years should have lessened most of the complications in the operation for senile cataract; but changed technique in the hands of the less experienced may and does bring added dangers. There is, in fact, no single step in the operation which may not be the subject of controversy. When one sees the many sutures used in some cases and the more or less complicated mechanism, he may look with some profit upon an old method in which the capsulotomy and iridectomy were made with the cataract knife while making the corneal section; and the lens expressed by finger pressure through the lids. The few instruments used and the few seconds consumed in the operation makes this method of more than historic interest.

Many of the so-called minor complications which occur in making the incision are mostly the result of inexperience. The beginner should, therefore, practice the incision and iridectomy on pigs' and kittens' eyes until he can hold the eye with the double fixation forceps in one hand and make the incision with the other without pressure on the globe.

One must early learn to appreciate the refractive displacement of the knife in the anterior chamber in order to make a proper counter puncture. He also should not use the larger knives. The 32 mm. Graefe knife is best for him.

Most operators now prefer conjunctival flaps. Sometimes there is considerable bleeding from these flaps and blood may be drawn into the anterior chamber. The operation cannot proceed without removal of the blood either by pressure or anterior chamber irrigation. This is not entirely devoid of danger, vitreous may be lost in this maneuver.

One of the most serious disasters is sinking of the lens into the vitreous. This is more apt to happen with the Barraquer suction operation. It has happened to me and I have seen it

happen to others. While the lens rises up again and can be removed with the loop, it is a most serious complication.

Prolapse of Vitreous — If the vitreous is fluid it will escape as soon as the incision is made. Here it would be well for the operator to follow the suggestion of Harold Gifford. He read a paper before this academy in 1920 "On Backing Out of Cataract Operations."[1] He reported a case of a man 48 years of age in which the fluid vitreous escaped with the incision in such quantity that he stopped the operation and dealt with the cataract later by several needlings, finally getting useful vision, which was maintained for five years.

I have under my care now a similar case in which I wish I had followed Gifford's advice.

My patient was 47 years old, was known to have had vitreous opacities for some time, but had had good results with glasses until beginning sclerosis of his lenses. This process had advanced to such a point in his left eye that his vision was reduced to less than 20/200, his loss of vision being due more to the irregular astigmatism in the lens than to opacity. The operation was performed November 26, 1933. In making the incision and after its completion there was such an escape of fluid that the globe collapsed. I removed the lens with the hook. After weeks of convalescence and a needling, a retinal detachment was found which has since been operated with slight success. It is almost certain that the sclerosing lens and vitreous opacities in the other eye are also accompanied by fluid vitreous. I expect to deal with it by repeated needlings, as suggested by Gifford, but dread the long period of time which it will require and the uncertainty of the final result. In reporting his case, Dr. Gifford said his patient had lost all sight in the left eye by some form of vascular degeneration 15 or 20 years before. I believe this to be significant of tuberculosis or focal infection. My case has definitely had antrum disease and I have not yet become satisfied that there may not still be ethmoid disease.

The loss of normal vitreous in the cataract operation is entirely a different matter. Lancaster[2] says normal vitreous does not come out of itself. Twenty-five years ago, Beard[3] gave squeezing of the lids on the part of the patient as the major cause. He also condemned over-cocainization. Now we know that improved anesthesia and akinesia have been the most important factors in prevention of vitreous loss.

I know of no bad results from the Van Lint method of lid injections and it is certain to prevent spasmodic contraction of the orbicularis if properly introduced. The injection must be made deeply. It does not, however, prevent the patient from moving his eyes about, which may occur in the nervous patient. If prolapse of vitreous occurs and there has not been a conjunctival suture previously inserted the eye should be closed at once. Any attempt to remove prolapsed vitreous or iris will result in more prolapse. If there has been a conjunctival suture so placed that it can be drawn up and tied with the lids closed, then some toilet of the wound may be possible.

One of my most serious cases of vitreous prolapse occurred just as the operation was finished. A nurse came suddenly into the room and asked me a question which caused the patient to turn his eye, with a serious prolapse of vitreous resulting. In the hospital operating room, quiet and freedom from distraction are essential. If there is a prolapse of normal vitreous before the lens is extracted, I believe it is better to stop the operation and remove the lens at a subsequent operation. For some reason, vitreous prolapse is not then so apt to occur. If one attempts to remove the lens with a spoon, unless he is very expert, he will lose more and more vitreous.

It is not alone the loss of vitreous, which if it is slight is not serious in itself, but the position in which it may leave the iris and capsule if they remain within the eye. This complication may result in an updrawn pupil or a very large pupil with the iris in the wound or retroverted due to the subsequent contraction of the vitreous, which has been in the wound and anterior chamber. Also, nearly always there are obstructive vitreous opacities. Thanks to Lindner,[4] we now know that vitreous is not a culture medium for the growth of bacteria.

Expulsive Hemorrhage—This accident has occurred but twice in my experience in the cataract operation, but several times in operations for glaucoma. One of the cataract cases I reported in 1931.[5] I did not then use retrobulbar injections of novocaine and adrenalin. In a series of cases of avertin anesthesia recorded by Miss Nichol, chief anesthetist at the Silver Cross Hospital, Joliet, the blood pressure was reduced in every case. Where it was high, 182, it was reduced to 100. Where it was low, 108, it was reduced to 66.

Dr. Davis[6] of Madison, Wisconsin, refers to a case of reduction of intraocular tension from 60 to 0 during avertin anesthesia. It would seem that this might be the ideal method to prevent hemorrhage and perhaps other complications when one can secure at once such marked reduction in general blood pressure and also in intraocular tension. If expulsive hemorrhage occurs, it is not advisable to remove the eye at once, as sometimes a normal globe may be retained.

In cases of intraocular infection, if discovered within 24 hours, most eyes can be saved by the deep orbital injection of 8 or 10 mm. of a solution of cyanid of mercury 1 to 1,000 with 4 mm. of 4 per cent cocaine added to prevent pain.

Very few operators think it wise to examine the eye the day after the operation. If they are so unfortunate as to have an infection they will probably lose the eye, as only early treatment is effective. The cases which I have lost from infection were those in which I did not examine the eye the day after the operation. Some foreign protein is useful in prevention and treatment. It is assumed that in every case the conjunctival sacs have had attention. There should never be any intraocular operation if the culture is positive.

I was recently informed of a case in which laboratory examination showed eleven colonies of staphylococcus growths. Notwithstanding this, operation was performed and the eye lost from infection. Any focus of infection about the face, mouth, nose, or ears should be taken care of. Lindner says he does not bother with a conjunctival culture previous to extraction.[7]

I once operated a case in which there had been a chronic conjunctivitis for several years. We were not able to free the eye from secretions and never secured a negative culture after treatment for two years. Finally, in the presence of conjunctivitis, with abundant secretion with bacteria of many sorts, extraction was performed. Daily intramuscular injections of 10 cc. of aolan were given and the patient made an uneventful recovery. We have no means of knowing when the patient may possess immunity or whether this treatment created immunity. However, at the time of the operation I frequently give injections of foreign protein.

No precaution should be omitted either as to patient, surgeon, or operating room. Is there any reason why the ophthalmic surgeon should not follow the rules of general surgery as to

dress, mask, and rubber gloves? The mask should cover the nose as well as the mouth. The surgeon will find on use that he can operate as well with rubber gloves as without. Only prejudice keeps us from making this advance, as I can testify from personal experience.

Intraocular hemorrhage occurring after the operation can be arrested by the subcutaneous injection of adrenalin and ergot. (Thilliez[8]).

Postoperative Glaucoma — A smooth intracapsular operation prevents this complication, because this condition is usually due to iris or capsular entanglements in the wound; also by cortex in the anterior chamber and to epithelial ingrowth into the anterior chamber. If the tension persists, some operative method is necessary; preferably an iridectomy or iris-inclusion operation or trephining. Glaucoma due to epithelial ingrowth is fatal to the eye.

This may be caused by a delay in healing due to the presence of iris, capsule or cortex in the wound or to the carrying into the eye of epithelial cells on the knife or other instruments introduced into the anterior chamber. The prevention of this condition is obvious. Leave no foreign substance in the wound and have point and edge of knife sharp and free from any roughness.

There are occasional cases of postoperative glaucoma (usually following needling), which may recover without operation.

Late iris and vitreous prolapse is usually due to some accident. A well-placed protective bandage and application of a Ring mask prevents this complication.

On December 18, 1934, I performed an intracapsular simple extraction on a man 68 years of age without complication, using one central conjunctival suture. Three days later a sudden cough caused him to have pain in the eye. Examination showed lateral prolapse of the iris, which was removed. As long as the central suture held, there was no prolapse above. However, a few days later this gave way and there was marked bulging and iris prolapse above. This caused slow convalescence and will require future surgery. I believe a firm pressure bandage might have prevented this complication.

Retinal detachment following cataract extraction is mostly incurable. It is not necessarily a sequel of the operation, but is caused by anterior choroiditis of tubercular or focal infection origin.

If an aphakic eye, in which visual results have been good, suddenly begins to become filled with vitreous opacities, treatment should be directed to whatever area of infection may be found. If none are discernible, then treatment with tuberculin is advisable. Fifty per cent of all uveal disease is tubercular. (Michael[9]).

The following is an illustrative case: The patient was 47 years of age. Two years after a perfect recovery from operation for cataract, retinal detachment occurred. This patient had been subjected to a most thorough physical examination. After a Walker diathermy operation attachment was secured which held for three months and then relapsed. The second eye was operated for cataract with perfect result. Two months following the extraction the vitreous in this eye became cloudy with exudate so that vision was reduced to 20/200. Metamorphopsia was also present. Under tuberculin treatment his vision returned to 6/5 with clearing of the vitreous and disappearance of the distortion. In this case, also, there was slight focal reaction several times following the injections. In such cases, I believe treatment with tuberculin is justified even though the skin tests may be negative, as it was in this case.

Irritation from retained cortex can only be prevented by its complete removal. If in any case cortex is present and not absorbing, the alternate use of atropine and eserine will help.

The surgeon may even be justified in the removal of cortex and capsule in a secondary operation; if necessary under a general anesthetic. I have had no personal experience in the use of lens proteins, but much experience in other foreign proteins, which I believe have their use in these cases.

I believe age is the greatest factor in the absorption of retained cortex; the older the patient the more the eye will be irritated by cortex. It is the common experience that lens cortex is well tolerated in the young.

Sympathetic Ophthalmia — This is fortunately a very rare complication. I once thought I had such a case until the pathologist reported that the enucleated eye did not show the characteristic epithelioid cells. Clinically, it was a case of sympathetic ophthalmia and the final result in the sympathizing eye was only enough vision for the patient to get about. One other case which did show the cellular changes followed the same course and left the patient with 20/200 vision in the sympathizing eye.

There are two rare but important postoperative complications which have received little attention in the literature: Corneal opacity, not striated keratitis, but a permanent dense central corneal opacity. I have had four cases. I know of no treatment for this condition, but if it occurred in one eye, I should use an undivided conjunctival flap in operating the second eye, preferably the Cermak subconjunctival method, which involves only a lateral slit in the conjunctiva. This might help in maintaining the corneal nutrition.

The other complication is a fistula which should be dealt with early by a conjunctival flap placed over the fistula after cauterizing the fistula and the under surface of the flap with nitrate of silver.

Observations and Conclusions — The cataract surgeon should know his patient as well as possible. Therefore, a proper preoperative general physical examination is most important and should not be slighted. Also the examination of both eyes, especially the pupillary reactions and intraocular tension should be known.

If one is operating in a strange environment or operates infrequently, he may operate on paper first. That is, make a careful list of his requirements and the various drugs and instruments which he will use, and go over this in advance with the operating room assistants.

Reserve the intracapsular operation for immature lenses in people over 55 years of age. Perform capsulotomy in mature cataracts of whatever age. In people under 55 years of age in immature lenses, perform capsulotomy removing cortex by pressure and anterior chamber irrigation, what is then left is usually taken care of by absorption without serious consequences.

I feel that the conjunctival sutures are an advantage, but are not a sure preventive of complications except that the upper lid cannot enter into the anterior chamber. This accident has occurred twice in my experience, but fortunately was discovered early enough to prevent loss of the eye.

Now the tendency is toward a peripheral iridectomy or iridotomy. This does not always prevent prolapse of the iris so that full iridectomy is still the safer procedure. The advantage of the round pupil is so great, however, that there are many cases in the middle years of life when it can be preserved without

prolapse if the wound is covered with a previously formed conjunctival sliding flap (the Van Lint flap).

I feel the need now is the perfection of a method for the removal of retained capsule in older patients. Arnold Knapp expressed this need in 1928,[10] asking for better illumination. Hildreth[11] recommends the ultraviolet light. Nugent[12] writes in favor of it and thinks the need has been met by the Hildreth light. I think the difficulty is not so much in seeing the capsule, except in certain cases, but in grasping it with any kind of forceps. May one hazard a prediction that some technique will be found by which this troublesome capsule may be removed after the lens is extracted? It is only exceptionally that one can remove the capsule with forceps. This need has not been entirely met by the instrument of D. M. Yazujian described in a recent issue of the American Journal of Ophthalmology.[13] Where the capsule is thickened, it may be removed by the capsule hook, but the thin capsule can be dealt with, with mild irrigation and mild suction.

Senile cataract is so common and so disabling to the patient and its removal so dramatic or so tragic as it may be, that we cannot spend too much time or thought on our failures as well as our successes. May I quote from Nicholas Senn:

"Brilliant operators are not always the best surgeons. The best results in surgery follow the one who is most painstaking in the minutest detail."

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DISCUSSION

Dr. John O. McReynolds, Dallas, Texas: After conference last night with Dr. Woodruff and some of our colleagues, it was considered inadvisable that I should undertake to touch upon ten or thirty points in his paper in the limited time of ten minutes. It was, therefore, regarded as best, if I could, to single-shot a single phase of his paper, and it was decided that that single shot should be directed to the undivided conjunctival bridge.

I shall not undertake to introduce any new principle in ophthalmic surgery, I am afraid that that day has passed, but I should be content if I could simply present some modifications of a well-established and ancient principle that will enable us to get a safer and more secure result.

I will try to explain in a few words how I try to make an undivided conjunctival bridge in a cataract extraction, which, by the way, is applicable equally to all forms of cataract and to glaucoma and other operations on the iris.

With a specially constructed conjunctival forceps, grasp firmly the conjunctival and subconjunctival tissue above the cornea, about 4 mm. above the limbus. This then gives us a vertical fold of conjunctival and subconjunctival tissue, and with the forceps in this position you have a perfect grasp of the eyeball so that it cannot move in any direction, and inasmuch as you are grasping the conjunctival tissue and subconjunctival tissue from above, you can exert a gentle upward traction on the globe, which lowers the tension within the eyeball instead of increasing the tension, as is usually the case when we apply pressure below the cornea. This is of importance in as much as it minimizes the tendency towards a falling forward of the iris across the blade of the knife during transfixion.

It is desirable, I think, to complete the conjunctival bridge with the knife alone, but if you are uncertain about this, it can easily be completed with scissors of the type I use, so they can be used in any desired direction, and in that way you can fashion the flap or the bridge in accordance with your wishes. I think about 10 or 12 mm. in breadth would be the appropriate flap.

In making the corneal incision with a knife, it should not extend more than a third of the limbus circumference. If desirable one may extend the excision on either side with scissors. That gives the gradual opening of the eyeball with no pressure on the eyeball, but, on the contrary, grasping it above and holding the eyeball down has a similar effect to a superior rectus suture and has the advantage, as pressure exerted on one point by the superior rectus suture may distort the eyeball to some extent and interfere with smooth delivery of the lens.

Now we come to the most important point. What are you going to do with your flap, or what are you going to do with your bridge? Recently, on a visit to Brooklyn, Dr. Jameson, who, by the way, has a marvelous hospital in Brooklyn and is using the conjunctival bridge, brought out prominently this point: Do not have the patient look down while you are undertaking to deliver the lens, because that tightens the conjunctival bridge. After the bridge is formed, I insert conjunctival grasping forceps,

one blade below the bridge and one above it, carry it back to the region of the insertion of the superior rectus so I have simply the bridge firmly in the grasp of the conjunctival forceps. Then if I want to relax this conjunctival bridge, all I have to do is to carry forward my forceps to the region of the cornea. Moreover, I have the bridge completely under my control at all times so that I can elevate it from the cornea or depress it or pull it back toward the cornea, or any direction I please. Therefore, if I wish to make an iridectomy, all I have to do is to lift up the bridge and make the iridectomy in the regular way, or if I want to make a glaucoma operation I still have the flap under perfect control.

What is the advantage of all this? Personally, I think it helps us to make secure the eye against many of these complications to which Dr. Woodruff has referred. I will say, in passing, that all of these various kinds of incisions have been attended with fine results. For forty years I have made every kind of incision and every kind of flap, and what I am presenting now is simply in order that we may have another string to the bow. When we undertake to make an operation for cataract, I think we want to be able to shift our position as often as may be necessary in the interest of the patient.

First of all, it gives immediate nourishment to the cornea. There is no interval of non-nourishment for the cornea. There are no stitches or sutures about the eye at all to give possible trouble, and when the conjunctival bridge is pushed back in position you have a perfectly smooth surface that unites, not tomorrow or the day after tomorrow, but immediately, and the anterior chamber fills immediately. You have the opportunity in that way for the surest and swiftest possible union. It is this swift union, with minimum of trauma, that helps prevent the prolapse of vitreous, that helps prevent infection, that helps prevent the accumulation of epithelial cells in the anterior chamber, that helps prevent prolapse. In other words, I think it is very effective in minimizing many of these complications we all so much dread.

I thank you very much for your kindness. I did not wish to present anything that had the appearance of claiming to be revolutionary.

Dr. Frank E. Burch, St. Paul: I would like to discuss one phase of Dr. Woodruff's remarks about chronic conjunctival infection. All of us have had the experience of sometime having intractable conjunctivitis which is very chronic and will not respond to treatment. In such cases I have on many occasions resorted to the so-called three-day treatment, two hours before operation instilling 2 per cent silver nitrate. This has proved very satisfactory in this type of case.

The other point I wish to discuss, as a means of preventing complications in cataract surgery, is to emphasize the importance of simplified operation. Many younger ophthalmologists feel that they have not reached the zenith of ophthalmic surgery unless they are invariably doing intracapsular extraction. To advocate a preference for intracapsular operations for the occasional operator, in my opinion, is to increase the percentage of hazard and the percentage of complications.

There is no gainsaying the fact that the intracapsular operation in the hands of the eye surgeon who is not operating very frequently and is doing less than one cataract operation per week is attended with an increased percentage of risk.

The classical extracapsular operation with capsulectomy permits a free opening in the capsule, permits lavage of the residual lens pulp, and permits results in operations upon immature cataracts which are practically equal in the end results, as regards vision, to those attempted by intracapsular methods attended with greater risk.

Based upon personal experience and observation, it is extremely doubtful that the operator who is not doing several cataract operations a week can offer to his patients the same assurance against loss of vitreous and other postoperative complications, or even the late complications and sequelæ when the supporting capsular barrier between vitreous and anterior chamber has been removed, as it is with the intracapsular operation. At the risk of being considered reactionary and old-fashioned, I am willing to go on record as being favorable to the classical extracapsular operation for cataract as the safest procedure for the occasional operator.

There is so much enthusiasm about intracapsular operation that many operators feel that they must do it. One of the arguments is that the frequency of needlings after the extracapsular operation is extremely important. I went over my own figures for a period of two years prior to January 1 of this year and found the percentage of needlings was 15 per cent, that the accidents of the needlings were only two cases in two years. One of these had detached retina and exceedingly high myopia, and the other had a severe iritis which was not explainable.

Following all the precautions as regards akinesia and use of sutures, a good capsulectomy or capsulotomy, combined with lavage, has afforded results in my own hands as regards the ultimate visual results which are equally good over a large series as compared with the figures offered by those who favor the intracapsular operation. Therefore, I believe the advantage of obtaining a clear pupil does not seem to weigh against the disadvantage of intracapsular method in perhaps 90 per cent of the eye surgeons who operate for cataract.

Dr. Hans Barkan, San Francisco: I intended to say something about conjunctival flaps, but after the beautiful exposition we have had I will not mention any little thoughts I have about them because they are not of sufficient importance. There are some complications, if you do not do a conjunctival flap, which might be mentioned from my own personal experience.

In doing intracapsular extractions and in putting in sutures we often find that the sutures, especially if we use more than one, irritate afterwards and the sutures are apt to fester and be surrounded by a drop of yellow exudate the third or fourth day. The patient feels disagreeable.

That can be obviated by using Mongolian hair. I learned that from Dr. Lindner. He used human Caucasian hair for the sutures. When he

saw my Japanese maid, he said, "Splendid! Mongolian hair is thicker and tougher and has advantages over all other hair for sutures. It is very fine, columnar and has no grooves or twists in which bacteria can collect." So since that time my Japanese maid has lost a number of hairs and I hope she loses a lot more in the future.

There is one point about these conjunctivitis cases before operating on them that I want to mention. I, too, have had, as we all have had, cases that have a positive culture and no matter what treatment is used the culture stays positive, even though the eye sometimes is clinically clean. You are apprehensive about it, and when it gets to a satisfactory state you don't want to be bothered about being apprehensive; it is too much of a chore. A number have cleaned up remarkably by blocking off both punctums with a toothpick applicator soaked in trichloracetic acid. It gets into the wood of the applicator and nothing runs off that wood. It soaks up any drop you may have. You put it into the two punctums. If you keep on treating your conjunctivitis by any method and abolish any connection between the nose, it gets better within two or three days' time before operation.

Another word about a complication Dr. Woodruff didn't mention and could not mention, just as little as he could mention all the complications inside a day's period. That is the dinitrophenol cataract. We in San Francisco started it; we had that dubious privilege. Dinitrophenol for rapid loss of weight was originated and taken up at Stanford Medical School by our Pharmacological Department. A year and a half ago the work was finished in the laboratory and the thing dispensed and used by a good many of our men in San Francisco. It was the quickest way for a fat, lazy, voracious person around the middle fifties to take off weight rapidly and not suffer any inconvenience.

We have seen seventeen such cataracts in one city. I have seen seven of them — four in the clinical material, three in private practice — and operated on three of them. They form a clinical group which simply has not appeared in our experience before.

Within a period of about two months, there appeared a group of people — in my own experience seven, in the total experience of the city seventeen — who are all about in the early fifties. They all happen to be women. They all had been very obese, and they all had taken dinitrophenol. They had all seemed well previously. They had all noticed failing vision within the last three or four months, and they all had cataracts, which in some cases were practically mature, in others so far advanced that they are maturing now, within the last two months or so, very rapidly. So as far as the clinical entity is concerned, there is no question that it is habit.

There has been some comment made about these cases by our pharmacological friends at home, who do not believe that dinitrophenol has had any relation to the development of cataract. I think there it is quite fair to take the clinical point of view. Here we have a group of people who have not appeared before under these circumstances, and they have all been taking dinitrophenol. It is up to you to explain why dinitrophenol has not conduced toward it in some fashion or other. Just what its action is, we do not know.

We have two very excellent men there now, one an associate of mine, the other a pharmacologist, trying to find out by every possible laboratory experiment just what it is that starts the process in the lens.

DR. OSCAR B. NUGENT, Chicago: The proper removal of cataract is an operation of the major type. The disasters, accidents and complications to which it is subject may be brought about by even the slightest deviation from the intended surgical technic, and because of this fact, I wish to stress the point which Dr. Woodruff has brought out — the importance of teamwork with well-trained assistants.

We have used the Hildreth lamp for more than a year and I cannot let this opportunity pass without again commenting on its value in the removal of the lens contents in case of broken capsule or in cases of capsulotomy removal of immature lenses in younger persons.

The character of the ultraviolet ray, produced by the lamp, causes a fluorescence of the lens cortex and nucleus, which readily enables one to recognize and remove the smallest portion of lens content which may still remain in the anterior chamber.

The more I use this lamp the better I like it, because the capsules which Dr. Woodruff has referred to as having been almost impossible to remove are readily removed under the influence of this light, because the capsule is more or less fluorescent and when you can see the difference between the capsule and the vitreous, as you cannot see it without the lamp, you know what you are doing and it is not difficult to go back and get some of the capsule that is left in.

Dr. Woodruff has mentioned that the intracapsular operation performed before the age of 55 is more difficult. My experience with the vacuum method of traction has taught me that even as young as 45 years of age is not a difficult age for delivery of cataract in capsule.

Contrary to the general trend of what the doctor has brought out concerning sutures, I have not been satisfied with one or two sutures in the conjunctiva and had acquired the habit of using as many as four or five. When I used five sutures, I recognized the benefit of more sutures, until I got up to using as many as nine sutures. Since last spring I have been using a modified untied suture, which I would like to show.

This is a double-armed silk suture, specially made. It is placed after the incision and before the removal of the lens.

DR. GEORGIANA THEOBALD, Oak Park, Ill.: At the Illinois Eye and Ear Infirmary during the past twenty years approximately 10,000 cataract operations were performed by the various staff surgeons and their assistants.

During the same period 51 eyes were enucleated and 10 eyes were exenterated for complications occurring during or as the result of the cataract extraction. The diagnosis and number of cases is as follows:

Loss of vitreous and iris prolapse	4 cases
Intraocular hemorrhage	13 cases
Panophthalmitis:	
Enucleated	5 cases
Exenterated	11 cases
Endophthalmitis	4 cases
Iridocyclitis	12 cases
(Some of these eyes have regressed to atrophic bulbi)	
Glaucoma	12 cases
Iris prolapse plus intraocular tumor	1 case

One private case sent to the laboratory had glaucoma secondary to a cyst formed by ingrowth of epithelium.

Thought should be given to avoiding the above results. Dr. Woodruff has covered the subject well; I wish only to emphasize some of his teachings.

Loss of vitreous and iris prolapse usually occur where the patient cooperates poorly. From the time of the first visit, the patient should be trained to obey orders; trained gently and firmly; he should be taught repeatedly to look down toward the feet, up toward the hair, and to hold each position until another order is given. After the operation, I have found it of great value to have my patient look up constantly, as while looking up it is impossible to squeeze and there is no tension on the flap. The patient should be repeatedly reminded to keep looking up. Hemorrhages occur usually before the first dressing and are due to friability of the vessels, lowered intraocular pressure, and thrombosis of various vessels.

Panophthalmitis is a needless occurrence. In the 16 cases mentioned here the operator proceeded in spite of the laboratory report of "dirty culture." Culture should be made on all cases and the operator should abide by the laboratory report.

Iridocyclitis is a more common occurrence and all such afflicted eyes do not come to enucleation. This kind of inflammation is due either to focal infection or allergy to lens substance.

Glaucoma occurs as the result of iridocyclitis. In all of these eyes there is a large amount of lens substance still remaining. Perhaps irrigating the anterior chamber or sucking out the lens fragments seen at the time of the first dressing might reduce the number of such cases.

Endophthalmitis is usually due to focal infection and such eyes are removed only when there is great pain.

Epithelium may be carried into the anterior chamber by a dull knife. Guard your knife point jealousy; do not entrust it to an inexperienced nurse.

In the one case I mentioned, "iris prolapse plus intraocular tumor," the history read that there was no light perception on the nasal side. Where light projection is poor, some fundus pathology must be anticipated.

A consultation with the family physician regarding the patient's health and habits is most necessary. He, too, can advise you about the preoperative sedative by mouth, which usually lessens the patient's fears and makes him more cooperative.

DR. RICHARD J. TIVNEN, Chicago: Dr. Woodruff has given us an exceedingly practical presentation of a very important subject. In the brief time allotted for discussion, manifestly, one may only refer to one or two of the great number of complications he has enumerated. The point I wish to stress is that more heed be given, as a routine measure, to the various pre-operative details.

Paramount among these is a thoroughgoing investigation of every physical detail — both systemic and local — pertaining to the patient himself.

As we know, from the moment the actual operation begins, until it is concluded, the development or avoidance of complications may be in great part an individual equation — that is, influenced by the operative skill, the experience, and the judgment of the operator. This is not, however, true of the pre-operative management of the case. Even the beginner in cataract surgery is almost on a par with the veteran at this period in the handling of the case and has almost equal opportunity with the veteran to devote 100 per cent attention to the various pre-operative details which will safeguard his patient.

The details covering the pre-operative investigation are well known to all of us and need not be discussed.

A number of exceedingly useful measures I have personally found of value are the following:

I always give my prospective cataract operative cases a number of "rehearsals" at my office before the operation. The patient is placed on a table, the eye cocainized, speculum introduced, and he is directed to move eyes in various directions; the speculum is removed, and he is directed to gently close his eyes, without squeezing. A bandage is then applied to both eyes and left on for five to ten minutes.

These "rehearsals," I have found, prepare the patient in advance for what is expected of him and perhaps contribute considerably in securing his later intelligent cooperation, a factor we know is of great moment in operative success.

I also operate the patient in bed, which is wheeled into the operating room, the head board lowered. I find this conduces to the patient's comfort, reduces the movements of the patient, and, in consequence, contributes to the maintenance of his psychic stability, all of which promote a successful operative issue.

Much also might be said of our ophthalmic surgeons insisting upon an adequate eye service in our general hospitals. Considerable improvement in this respect is evident, but we still have far to go. Only continuous insistence on our part can bring about the desired improvement.

DR. S. B. MUNCASTER, Washington, D. C.: I have been experimenting to find some way to prevent the bright lights shining upon the instruments while performing an operation on the eyes. I found that No. 3 soft light ground in your corrective glasses for correcting your vision will be of great benefit. No. 4 is too strong and you can't see as well, and No. 2 will not be enough. No. 3 will help you in removing this bright light from the eyes.

Also in some cases I have had some of my instruments made in a dull finish so as to prevent that light from shining direct to the eye. Sometimes it is almost dazzling to a sensitive eye.

Dr. J. G. Huizinga, Holland, Mich.: In getting rid of all the lens débris after the ordinary capsulotomy operation, I have found a great deal of help in obtaining as large as possible a capsule fragment. I find that I get this largely through two perpendicular incisions through the capsule with an exceptionally sharp, new cystitome. We all try to have our knives as sharp as possible. We fail frequently because we do not have a cystitome that is equally sharp. By making two parallel perpendicular incisions through the anterior capsule, I am able, with my forceps, to grasp an exceptionally large part of that anterior capsule and draw it out, leaving less of a pocket in which the lens débris may accumulate or reside.

There is another matter which has not been mentioned, in which I have compared my results recently with those which have been done previously, namely the old thing that has been so often mentioned in the books, but which we so frequently ignore, the preliminary iridectomy. That is up to you. If you do an iridectomy at all, do it a few weeks in advance of the time you are expecting to do your cataract operation. I am sure there are not many men here who, if they had seen the results of that method, would choose on their own eyes to have anything else done but a preliminary iridectomy. It makes the subsequent cataract operation so much simpler, so much easier, so much less trouble in the way of irritations, and so much less danger from infection, that it is well worth our while to tell the patient of it. I find no patient of mine has ever refused the preliminary iridectomy, because after I have explained the advantages of it they are entirely willing to have it done.

The third thing I wish to call attention to is this: As a result of rather sad experience, when I formerly allowed my assistant to take care of some of these cases after I had done my operation, I found that no one, at least in my office, should take care of these cases after the operation but the surgeon himself. Anyone else interfering with that or doing the dressing on that patient is taking a tremendous chance of undoing the good work that the surgeon has done.

Dr. Harry W. Woodruff, closing: I said in my paper there is scarcely any part of the cataract operation or any suggestion or any change in procedure or technic that is not subject to controversy. Many of these that have been brought up are as old as my experience in the cataract operation. We used to discuss those questions when I first began to operate, and they are not settled yet.

It was interesting to see Dr. McReynolds' moving picture of the intracapsular operation under the conjunctival bridge. I have considered that subconjunctival operation, but I feel that, while it may be of value

in Dr. McReynolds' experience and in the experience of others, it would be only in a case where it was necessary to preserve the nutrition of the cornea on account of having had the experience of the development of a general opacity in the cornea of the other eye. Then I would think that a complete subconjunctival extraction — that is, preserving all the conjunctiva, not simply a bridge, but making enough of an opening in the conjunctiva to make the incision and to get the lens out — would be the procedure. I don't know how much good the bridge would do — a comparatively narrow bridge — in maintaining the nutrition of the cornea. It would do some good, of course.

Dr. Burch refers to nitrate of silver for preparation in conjunctivitis cases preliminary to extraction. Of course, I believe in nitrate of silver absolutely. In obstinate cases of conjunctivitis I use much stronger solutions. The patient I referred to in my paper had all those treatments, week after week, and still nothing would cure the conjunctivitis, either clinically or bacteriologically.

Whether the administration of foreign protein took us safely through this operation, I am not so sure, but I know there is another element besides the local condition of the conjunctiva. This I can illustrate in a case of iridocyclitis, pus pouring out of the lacrymal sac and atrophy of the conjunctiva in a case of trachoma, with the eyelashes running on the cornea so the epithelium was abraded through a considerable area. Here was an eroded epithelium, pus coming out of the lacrymal sac, bathing it, but no apparent infection of the cornea for weeks. So there is some other factor besides the local condition, and maybe you can supply it by foreign protein in some cases.

I have had more than one discussion about the needlings and capsulotomy cases. I know that most authorities talk about the few cases in which needlings are necessary, but that isn't so in my experience. They are too frequent. I have to do so many of them. I don't know that I can tell why that is. I feel, however, that in many cases that you refract and get a good visual result, you still have the posterior lens capsule, which later on is going to make you trouble. It is bound to wrinkle, and if you have a posterior lens capsule that is visible on oblique illumination or with the Hildreth lamp, which has been referred to, I believe you had better needle it shortly after your operation rather than wait a year or more.

Dr. Barkan refers to Mongolian hair. Of course, he lives in a country where you can get those hairs comparatively easily. In our part of the country we don't have any Mongolian hairs. However, I have often felt that irritation in there from those sutures was an important factor and if we could get rid of it, it would be advantageous, of course. I heard Dr. Lindner refer to the use of the Japanese women's hair as supplying that want.

I have never had any experience with cataracts produced by dinitrophenol. Of course, that is not any part of this paper, the causation of cataracts, so I will pass that up.

Dr. Nugent still talks about removing the capsule with the forceps, and I won't deny his ability to do that, but I admit my own inability

to get the capsule with the forceps, only in exceptional cases. I have been able to do it, but I cannot do it as easily as it looks as though it could be done. The capsule eludes the forceps. That is the reason I made that reference to some kind of hook that you may be able to get that with.

Dr. Nugent also made the statement that when the patient is as young as 45, the intracapsular operation can be performed by the vacuum method. That might be all right for Dr. Nugent, but it would not be all right for the ordinary operator. He has to be conservative. He can't see how far he can go to the edge without getting over; he has to keep back and be on the safe side. We do know positively that the suspension is more fragile in old people than it is in young people, directly in proportion to the age.

He also mentioned many sutures. Dr. Nugent can put in just as many sutures as he wants to, but that statement does not apply to the average operator; he should not put in all those sutures.

I have tried the untied suture, not the one Dr. Nugent describes, but one Dr. Berens of New York published some time ago. While perhaps if I had more experience with it I might think of an advantage, I haven't yet succeeded in any kind of an untied suture to fill the requirement in the operation of cataract.

Dr. Theobald says panophthalmitis is unnecessary. Well, a lot of these complications are unnecessary, of course, but that is the thing we are aiming at, to perfect our technic. I believe that an infected eye following a cataract operation is a pretty serious criticism of the operator.

With reference to general hospitals, if the hospital does not have everything in the operating room that the operating eye surgeon thinks it should have, it is up to him. People will have those things if they know about them — that is, most hospitals are ready and willing to go along with their surgeons and get and have what they want. Very often it is too much bother; you only have an occasional case. I think it is tremendously important. As Dr. Tivnen says, this operation is built up from a number of small things and any one of those little items may mean the failure of your operation.

I never had any experience with any complications on account of light being reflected, as Dr. Muncaster has referred to.

Preliminary iridectomy has been discussed for years. I believe there are occasional cases where it is an advantage, but I am quite sure that the majority of the operators would not advocate preliminary iridectomy as a routine procedure.

Regarding the assistant's taking care of the cases afterwards, I would say that is a personal question. You may have an assistant who would take care of them better than you would. Perhaps he can do the operation better than you.

ANISEIKONIA

WENDELL L. HUGHES, M.D.

HEMPSTEAD, L. I., N. Y.

ANISEIKONIA is the condition of incongruity of the size or shape of the ocular images. Its existence under certain circumstances has long been known, particularly where the lens of one eye has been removed or in cases of antimetropia, or anisometropia of high degree. Until recently it has not been recognized that small inequalities of the images may be of extreme importance and that these asymmetries may exist where the refraction of each eye is the same and frequently when there is no refractive error in either eye.

Credit for bringing out these facts belongs to the exhaustive and exacting efforts of the Department of Research in Physiological Optics of the Dartmouth Medical School, where excellent work has been done in devising methods of measuring the small defects and designing lenses to correct the inequalities found.

When the higher amounts of aniseikonia exist, fusion of the two ocular images into one is impossible and either suppression of one image or diplopia must exist. Image disparities of less than 5 per cent may be fused, but with more or less difficulty, depending on the amount of asymmetry present. Many cases of definitely proven aniseikonia present no symptoms, just as many cases of other refractive errors, such as astigmatism, hyperopia, and hyperphoria, give the patient no discomfort.

There are, however, many cases which have severe symptoms. The most common ones, in the order found in our series of 350 cases, being:

1. Eyeache and headache
2. Photophobia
3. Indefinite ocular discomfort on use of the eyes, and the general asthenopic symptoms found in the more commonly recognized refractive errors.

When ocular discomfort or headache on use of the eyes persists after correction of the ordinary refractive errors, aniseikonia should be suspected. Particularly, the presence of photophobia, not accounted for by diseased conditions, should make one suspect the presence of this particular defect. Many, if not most, patients who find comfort in tinted lenses have aniseikonia as the cause of their discomfort. It has been found that the correction of the aniseikonia relieves the photophobia and tinted lenses are very rarely necessary.

One type of headache, in which the correction of aniseikonia has been unusually effective, is the unilateral, frequently recurring and often prostrating headache, usually coming on without definite relation to use of the eyes and often accompanied by nausea and photophobia, the latter not particularly limited to the time of the attack. This is often diagnosed as migraine. In few of the cases that have been helped has there been any premonitory aura.

Case Report — Dr. L. F. complained of very severe prostrating headaches, recurring at frequent intervals, the attacks usually lasting two or three days. Examination of the eyes showed considerable hyperopic astigmatism corrected by the following formula:

O.D. S. $+4.50 \supset$ C. $+1.75$ Ax. 75°

O.S. S. $+2.50 \supset$ C. $+2.25$ Ax. 108°

Visual acuity with glasses equalled 20/15 O.U. In addition, there was a right hyperphoria of 1.25 $\triangle$. Inasmuch as there was not a great change in the refractive correction found at the examination and the lenses he had been wearing, it was suggested that the relative size of the visual images be measured.

Examination on the ophthalmo-eikonometer showed the right image to be 0.75 per cent smaller in the vertical meridian than the left image, and the left image to be 0.75 per cent smaller horizontally than the right image.

Iseikonic lenses were prescribed to correct this inequality of the visual images, and since wearing the lenses he has had no headaches and is able to go about his practice with comfort.

On the suggested possibility of aniseikonia being a factor in the production of true migraine, a special series of 28 cases was examined in cooperation with Dr. O'Sullivan of the migraine clinic at Bellevue Hospital, New York. Only a small portion had aniseikonia, in fact, two of the cases occurred in one-eyed individuals. Aniseikonia existed no more frequently in these patients than in any other group of cases chosen at random.

The relation of aniseikonia to various phorias and tropias has not as yet been definitely worked out, but it is a fact that cases that present aniseikonia very frequently have small phorias. It has been observed that in many cases presenting hyperphoria, it has diminished or completely disappeared on the correction of the aniseikonia. It has been our experience that prisms are very rarely necessary when aniseikonia is present and properly corrected. It has also been found that most cases of strabismus in which the size of the images can be compared have had quite a high degree of inequality of the visual images. If the difference is greater than 5 per cent, the images cannot be fused. This asymmetry of the images is a cause of the persistent diplopia, occasionally occurring after squint operations, the so-called "horror fusionis."

This was graphically demonstrated in the case reported by Professor Bielschowsky early this year. By means of the correction of a disparity of about 13 per cent in the size of the images, the diplopia immediately disappeared and excellent depth perception was proven by measurement with the Keystone Stereometric cards. On removal of the lenses, cyclotorsional diplopia was present. A most surprising phenomenon was that binocular single vision was maintained not only in the central portion, but throughout the entire field of binocular fixation.

Method of Measurement — The apparatus has been fully described in the publications of the Dartmouth group. The patient looks through the size-measuring instrument called the eikonometer at a screen with four small holes, 4° from the center, with lights behind these holes so arranged that one eye sees the holes as black dots and the other eye sees the lights only. The dots and lights, coming from exactly the same situation, normally are apparently superimposed. When there is esophoria, exophoria, or hyperphoria with no aniseikonia, there is displacement of the lights in relation to the dots, the relative vertical and horizontal dimensions remaining the same. The four images seen by the right eye (lights) are shifted to the right or left or up or down in relation to the corresponding dots. Lenses for the correction of this inequality must be very carefully calculated from tables (specially prepared by the Department of Research in Physiolgical Optics at Dartmouth College) as to their curvatures and thicknesses, and be very carefully ground to produce the desired result. Frequently a double lens must be designed.

This may be of the double eyewire or temporary type, or the lenses may be designed as in the so-called permanent type to fit in one eyewire.

In general, for plus power lenses the more highly curved the inside surface is ground and the thicker the lens, the greater the magnification produced. Still more magnification and meridional differences in magnifications can be obtained by combinations making up telescopic systems to give the necessary spherical, cylindrical, and iseikonic corrections.

Results — The results here recorded are limited to the first 250 cases examined. This is done to exclude the ones for which lenses have only recently been prescribed and those which have not had their lenses a sufficient length of time to definitely determine the results. Practically all of these patients were referred by ophthalmologists, because they were suspected of having aniseikonia.

Of 357 cases referred for examination, aniseikonia was found in 235 or 65.8 per cent. In 191 cases, the patient obtained iseikonic lenses and was rechecked after wearing the first or temporary pair for varying lengths of time, usually at least three weeks, before the permanent prescription was given. The findings were then rechecked. All cases included in this group have had their lenses over four months with the following results:

1. 89 or 46.6 per cent obtained complete relief
2. 50 or 26.2 per cent obtained marked, though not complete relief
3. 5 or 2.7 per cent obtained only slight relief from the temporary glasses, usually insufficient to warrant having the more expensive single eyewire type made up.
4. 47 or 24.5 per cent obtained no relief

Discussion — A means of helping approximately 75 per cent of the difficult refraction cases whose symptoms had not been relieved by the ordinary refractive methods has been applied clinically in this series of cases.

Drawbacks

1. Length of time required for the examination and, occasionally, uncertainty of the results.
2. Expense of the instruments to the examiner.
3. Expense to the patient for the lengthy examination and for the specially-designed glasses.

Despite these drawbacks, however, if relief of the distressing symptoms of even the majority of this difficult class of patients, who have usually been examined many times without relief, can be obtained in this manner, our efforts have not been in vain.

Another window has been opened to admit light on many heretofore poorly-understood phenomena. A great deal of further work will need to be done for their more complete elucidation. We know, for instance, that many cases of photophobia and hyperphoria are relieved without tinted lenses or prisms by correction of aniseikonia. As the new chapter is being written, how many of our present perplexities will be solved we know not. Let us advance with an open mind, alert to interpret and apply the new knowledge where it will do the most good for suffering humanity.*

DISCUSSION

Dr. Walter B. Lancaster, Boston: The subject of aniseikonia (its nature, measurement and treatment, and the results to be expected) has been before the profession for some years. The subject has been presented from a theoretical or physiological point of view showing how it affects binocular vision, space perception, the horopter. Clinical reports have been presented showing the results of correcting aniseikonia. But not much has been written by ophthalmologists about the subject. This is the first report on aniseikonia before one of the large societies by an ophthalmologist not connected with or working at the Dartmouth Clinic. One or two clinical reports have been published by optometrists.

It seems to me rather an important occasion. The time is opportune to make some sort of an appraisal of the work to see where we stand or where we ought to stand.

I have said that little has been written by ophthalmologists about aniseikonia: the reasons are that in the first place it involves a knowledge of physiology and of optics which few if any of us possess, and, therefore, few are competent to discuss it scientifically and from practical experience. In the second place the apparatus needed to measure aniseikonia has not been available to ophthalmologists in general, and if it were they could not use it satisfactorily without special training.

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* Full credit for the development of this work must be given to the Department of Research in Physiological Optics at Dartmouth Medical School. I am indebted to Professor Webb W. Weeks of the New York University and the New York Eye and Ear Infirmary in whose department this work was done; also to Mr. A. Haustetter who so graciously supplied the apparatus, and particularly to H. M. Fisher, O.D., who has so well conducted the majority of these exhaustive examinations. The cooperation of various ophthalmologists of the district is also gratefully acknowledged.

But if little has appeared in print, there has been considerable comment passed from mouth to mouth. Some of this has been favorable, of course, for the results have compelled recognition; but there has been a tendency in certain quarters to belittle the significance of aniseikonia and to throw doubt on the claims made for its treatment. I wish to discuss this side of the subject.

Dr. Hughes has given figures showing his results; Dr. Doane has reported his results, and the figures are very nearly the same. The figures published by the Dartmouth Clinic are about the same — say 70 per cent of cases benefited. The Dartmouth workers sent followup questionnaires to all of their cases.

Anyone who has any knowledge of the statistical study of results of treatment knows the sources of error to which it is exposed. It is necessary to discount the statements of patients as to results. It is necessary to allow for the profound psychological effect of prolonged examinations with an impressive instrument by an enthusiastic examiner. It is necessary to allow for possible bias on the part of the observer. When all these allowances have been made, what about the residue? How shall we account for cases which cannot be explained away? I am reminded of Dr. Warren's remark when he had just completed the first surgical operation under ether: "Gentlemen, this is no humbug."

I wish to point out certain very obvious reasons for the failure of eikonic lenses to relieve some patients whose symptoms strongly suggest that their trouble is, at least to some extent, due to aniseikonia. Let me compare the problems of aniseikonia to those of astigmatism. Suppose astigmatism were just discovered. Suppose apparatus were available to measure only the vertical and horizontal meridians and to correct the differences in refraction in those two meridians only. I think you will admit that much could justly be claimed for the treatment of astigmatism, even if we were thus limited. But it is certain that there would be many failures, namely: those cases with axes 20° to 40° away from the vertical or horizontal axes. Should we, therefore, decide that astigmatism was of negligible importance, not worth the attention of clinicians? Suppose I have an object whose size and shape are unknown to you. I permit you to measure its height vertically and its length horizontally — how little you then know its true dimensions! At present the eikonometer measures only the vertical and horizontal meridians — the most important beyond a doubt — but not the only ones worth considering. Corrections in oblique axes are being made occasionally at the laboratory at Dartmouth, but apparatus is not yet available for general use.

Furthermore, it must be borne in mind that we do not know how the eyes compensate for aniseikonia. We know that for hypermetropia the accommodation gives effective compensation, and its capacity to do so is measured by the *amplitude* of accommodation. We know that compensation for differences in brightness (in intensity of light) is effected by the pupil, and by retinal adaptation. We know that exophoria, for example, is compensated by convergence and we measure the *amplitude* of convergence as a gauge of this function. We know also that there is a capacity to compensate for aniseikonia, and can measure its *amplitude*.

We know that when the amount of aniseikonia is over about 5 per cent, the eyes, unable to compensate so large an amount, avoid the dilemma by suppression. But of *how* the eyes compensate for smaller amounts we are in complete ignorance. Lacking knowledge of this important element, it is not surprising that baffling observations are encountered, and problems still insoluble frequently arise. This is far from being a reason to reject the whole proposition.

Since we do not know how aniseikonia is compensated, we do not know just how it acts on the mechanism which takes care of refraction, and on the mechanism for binocular vision which takes care of heterophoria. But ignorance of *how* it works does not mean that we do not know that it *does* affect binocular vision and errors of refraction. This must be accepted, and further light awaited. It will come!

Another hitherto unconsidered factor was brought forward by Friedenwald at the last meeting of the American Medical Association. Anisophoria must be taken into account, and if uncorrected will explain some of the failures to relieve symptoms which it seems ought to be relieved. Professor Ames has designed an apparatus for accurate clinical measurement of anisophoria, and due weight will be given to it and additional light will surely be thrown on the whole subject.

But I submit that in trying to arrive at a sound evaluation of aniseikonia, we must not place too much weight on *clinical* results. Indeed, my own clinical cases show much less than 70 per cent of good results, partly because I have selected for trial more difficult and less favorable cases. This is shown by the results not only of my own treatment of them with eikonic lenses, but by the results obtained at Dartmouth on the cases I have sent them for treatment. Their percentage of successful results with these is much smaller than their general run of cases. Secondly, my results are less favorable than theirs because I am less expert and less experienced in the use of the apparatus.

Strong and convincing as are the clinical statistics which Dr. Hughes has laid before you, I believe that the scientific or, if you will, theoretical arguments are even more convincing.

The foundations of this research in aniseikonia are in unassailably sound optics and visual physiology. Due regard has been given to preceding work in the field by the recognized highest authorities, mostly German, and too little known in this country. The fact is important that Tschermak and Bielschowsky, the leading authorities in that special part of visual physiology, are well acquainted with the work of Ames and his staff, and freely acknowledge that the Dartmouth workers have extended their researches beyond the limits reached in Germany. It is always significant when those special workers who know most about a subject and have contributed in important ways themselves approve and endorse the research work of another group.

Again, the results by one method of study have been checked by other methods. Many of you have seen the tilting plane, shown at the meeting of the Association for Ophthalmic Research, which demonstrates so convincingly the effect on the horopter of aniseikonia. Many different

methods of checking the horopter have been used by Ames and his staff and mathematically analyzed.

The thought I wish to leave with you is that here is a large and important field of research. Only the surface has been scratched. Much digging is needed to uncover the whole truth. Do not be so prejudiced, narrow-minded, shortsighted as to reject or belittle the whole proposition just because it is incomplete. Rather give encouragement, support and praise to the pioneers.

DR. WENDELL L. HUGHES, closing: I think Dr. Lancaster has brought out some very important points, and I do agree very strongly with him that only the surface has been scratched in this new work. It is new work, and it is very difficult to evaluate exactly what is being done. We don't know where we are going. Each day we find out something new.

In regard to the relationship of aniseikonia to the various phorias, we don't know why in some instances a hyperphoria disappears with the correction of aniseikonia. The patient mentioned in the paper above with his corrected lenses, measured with Maddox rod, had no hyperphoria. Without his aniseikonic correction, he had a hyperphoria of one and a quarter degrees.

Further analysis of types of hyperphoria is being taken up now and there will be more to report on this subject later. Just which types of hyperphoria are corrected by this aniseikonic correction and which are not, we do not know at present.

CONGENITAL PARALYSIS OF THE SIXTH NERVE

JAMES WATSON WHITE, M.D.

NEW YORK

THERE WOULD seem to be no justification for bringing this subject to your attention were it not for the fact that incorrect diagnoses often lead to unsuccessful results from operative procedures.

The main symptom, that of limited or complete failure in abduction, is too well known and too obvious to mention, were it not for the fact that other conditions than a paralysis of the sixth nerve often appear as such, and for this reason the mistaken diagnoses occur.

An eye may be limited in abduction because of paralysis of the sixth nerve from any cause, such as birth injuries, or because of some anomaly of the muscle itself, such as a fragile lacy tendon which is sometimes found. In these cases, when the muscle proper is grasped by hook or forceps through a conjunctival incision, and the patient asked to look toward the field of the externus, the contraction of the muscle is often quite normal, but the effect is limited because of the character of the tendon; if the sixth nerve is involved, the contraction is absent or limited.

Again, when the sixth nerve is paralyzed, the internus of the same eye (or direct antagonist) usually develops a more or less marked secondary contracture, while, if or when the affected eye is used for fixation, the internus of the fellow eye (associate antagonist), has a secondary deviation, which is usually quite marked. These can be measured by the screen test, if the paralysis is not complete, and exaggerated by the screen-comitance test, when first one eye, then the other, is made to fix in the six cardinal fields, while the action of both eyes is observed by the examiner.

Either or both interni may become markedly over-developed, causing the affected eye to constantly remain at the inner canthus or each in turn to have a marked inversion when the fellow eye fixes.

The conditions for which paralyses of the sixth nerve are most commonly mistaken are a large powerful internus with a normal externus, a strabismus fixus and a retraction syndrome; and, indeed, in the first two conditions especially, it is often impossible to make a definite diagnosis until the muscles in question are explored and studied.

The feel of the muscles when on the hook is quite diagnostic and the sensation most like it is that gotten when fishing, the normal muscle feels as a strike by an ordinary fish; the large hypertense muscles, like an especially large fish, and the fibrous bands found in strabismus fixus and retraction syndromes like hooking a sunken log or reef. These sensations are rarely affected by an anæsthetic, except when the muscle is normal.

Examination

The deviation should first be measured for distance and for near, and an attempt should be made to measure in the six cardinal fields. This may not be possible in the field where abduction is absent or limited, but the attempt will often bring out primary and secondary deviations.

The screen-comitance test is most important here to bring out the primary and secondary deviations and also any complicating vertical anomaly. It may also show that when the fellow eye is covered and the supposedly paretic eye is required to fix, that the eye may be abducted normally or nearly so. The fellow eye should be watched carefully for secondary deviation, as the diagnosis and operative procedure may be governed by this observation. It seems, at times, that the effect or lack of effect on Tenon's capsule and the conjunctiva has some influence in determining the diagnosis. In examining children, one must have considerable patience. Often a change of test objects and procedure is necessary to hold the attention. With patience, however, the examination in children can be as satisfactory and the diagnosis as sure as in adults, except that in adults the secondary deviation and contracture may be increased to such an extent that they become more evident.

Paralysis of the external rectus may show simply a failure in abduction, or it may be complicated with such a marked secondary contracture that paralysis is more apparent than real, and when the internus is freed by tenotomy the externus may be found to be nearly normal. Then again, a congenitally large, powerful internus may be so strong as to keep the eye in the

nasal field so constantly that the externus only appears to be paralyzed. This was so in the following case reported in a previous paper.

Case I — C. B. was born with the right eye converged. At nine months of age, when I first saw him, the eye seemed anchored in the nasal field. The father, who was a doctor, observed him carefully from birth and had never seen the eye leave the inner canthus. Possibly some movement of the conjunctiva or Tenon's capsule made it seem that the externus would abduct the eye if it could, but at no time could any abduction be observed. The deviation by corneal reflex tests measured about 80 degrees.

On operation, at the age of 20 months, the internus was found to be very large and had the feel of a mighty fish. A recession was made to the equator. On the second day after the operation, when the first dressing was made, the eye could be abducted to the outer canthus and adducted to the inner canthus. This was three years ago and examination recently shows no deviation for distance or near nor in looking to the right or to the left.

A question arises here: How many of our disappointments in operations may be due to placing the cause in such a case to the externus and resecting this muscle without a recession, or with an insufficient recession of the internus? This is the most marked case the writer has observed, but there must be gradations of powerful interni and paretic externi. If such a condition were left unoperated, would the externus not become weakened by the constant pull of the powerful internus? This probability is advanced as an argument in favor of an early operation where such a condition is suspected.

Case II — H. B., age 14 months. There was a definite history of squint at 11 days after birth. Both eyes were in the nasal field, and apparently unable to abduct to the midline. A note from the history says, "much like C. B. (Case I), but bilateral." The parents were advised to patch each eye alternately for one week at a time and to practice abduction in any way possible. When seen July 19, 1935, there was still a convergent strabismus present, but each eye in turn could abduct to the outer canthus with some difficulty. The case thus appears to be one with hypertense interni, which relaxed somewhat due to the prolonged occlusion. The case looked much like a paralysis of both externi in September, 1934, but with the improvement in abduction, the original diagnosis seems the more probable. The treatment is to be continued for another six months before re-examination.

Cases of strabismus fixus appear very much like the case just described. The eye is fixed in the nasal field. The effect of traction on the conjunctiva and Tenon's capsule may be the same as in the powerful interni. There may be some evidence of giving of the internus in the latter, while in strabismus fixus, the eye is firmly fixed at the inner canthus. The head is rotated to the right and left depending on which eye is chosen for fixing. It is often quite difficult and even impossible to make a definite diagnosis without exploring the muscles.

In strabismus fixus, the internus is of the same fibrous construction as is found in a retraction syndrome, except, in the latter, it is usually of normal length, while in strabismus fixus it is a short rudimentary band, entirely fibrous, which holds the eye firmly in the nasal field. To get the band on a muscle hook, it is necessary to go well back and when so engaged no amount of traction in safety will bring the eye from this position. A case has been reported in which the globe was ruptured from such an attempt. As the externi in these cases are normal, a complete tenotomy would allow the normal externus to abduct the eye abnormally and finally to develop a secondary contracture with a resultant divergence. To ward against this, a generous recession of the fibrous internus should be made to or beyond the equator, using the fibrous band as a stay rope to prevent the eye from abducting too far.

Retraction syndromes when atypical may resemble very closely paralysis of the externus. There is no mistaking the typical case of retraction syndrome with limited abduction and limited adduction, each being dependent on the amount of fibrous tissue present, narrowing of the palpebral fissure in adduction and often in the primary position, retraction of the globe in adduction, the up and down shoot also in adduction and the remote convergence nearpoint.

It is not the purpose of this paper to mention any of the many typical forms, except those which resemble paralysis of the externus. In these, abduction may be nil or nearly so, but adduction may be normal; narrowing of the fissure may be slight or absent and retraction of the globe may appear to be absent. With the following test a retraction of the globe may be demonstrated in many cases in which it seemed to be absent. The tips of the index and middle fingers of one hand are placed one on each lower orbital margin, just opposite the origin of the inferior oblique. Making light but equal pressure with each finger, the

patient is asked to look right and left. The affected eye will be seen to retract from the lid in adduction, while the sound eye will approximate the lid throughout the excursion. In bilateral cases, each in turn will retract. With this test, retraction can be demonstrated in most cases and is the deciding point in some cases which appear first as simple external rectus paralysis.

The following cases will further demonstrate some points of difference and the pitfalls in diagnosis.

Case III — In a child age 20 months, both eyes were at the inner canthus with no abduction demonstrable. The condition was diagnosed as bilateral hypertense interni, and operation proved this to be so. The result was most satisfactory. However, in *Case IV*, (Jim, age 2 years) which appeared to be identical, there was found at operation bilateral fibrous internus and externus. This was an atypical retraction syndrome.

Case V — A patient 22 years of age had both eyes in the nasal field with no abduction. The face was rotated to the right and left to enable each eye in turn to fix. There seemed to be a slight narrowing of the palpebral fissure and retraction. Diagnosis: atypical retraction syndrome. At operation, the externus of the left eye was first exposed and was found to appear and feel like a normal muscle. The internus was then explored and found to be a short fibrous band typical of strabismus fixus. It was receded to the equator and the some procedure followed on the right eye, except for the preliminary exploration of the externus. Neither externus was resected and the cosmetic effect is quite satisfactory.

In a recent group of four cases presented with a diagnosis of paralysis of the externus, two had definitely powerful interni, one an atypical retraction syndrome. In the fourth it was impossible to determine whether an atypical retraction syndrome or a true externus paralysis was present. Exploration would definitely determine this, or further examination might have given further evidence.

Operation

In operating definite cases of paralysis of the sixth nerve, the writer prefers to advance the affected muscle to obtain all the mechanical help possible. This is supplemented by a recession of the direct antagonist, the internus of the same eye, to lessen the secondary contracture. When the internus of the fellow eye has a marked secondary deviation, a recession of this muscle is

also often necessary to lessen the secondary deviation. These procedures chosen carefully give satisfactory results. When operating either or both interni the convergence nearpoint must be carefully recorded. With the operation of taking a tongue from the superior and inferior recti and suturing to the externus in various ways, the writer has had no personal experience, except to examine several cases postoperatively. Had the results so obtained been gotten without a recession or tenotomy of the internus of the same eye, the writer would have adopted the procedure long since, but the internus is generally weakened at the same operation as the splicing. This may be a large factor in the results obtained. One must also respect the elevators and depressors and consider the chances of adding a vertical element to what was originally a simple lateral squint, which could be controlled by a turn of the head. We are all familiar with the weakened superior recti and the consequent diplopia following some of the operations for ptosis involving the superior rectus.

Conclusions

Experience leads one to make the diagnosis of paralysis of the sixth nerve with due regard to the conditions which may simulate it and to employ any means and a due amount of time when necessary to make the diagnosis.

At operation, one may have to change the diagnosis after the muscles are exposed and examined and so change the operative procedure.

DISCUSSION

Dr. John F. Gipner, Rochester, N. Y.: It is very easy for the ophthalmologist to fall into the error of regarding most ocular abduction deficiencies as paralyses of the external recti. We are grateful to Dr. White for emphasizing the muscle syndromes which simulate this condition and for giving us his methods of differentiating them from external rectus paralysis and from each other.

In my experience, an external rectus paralysis suitable for operative correction is rarely seen. Practically all the external rectus paralysis cases seen in our clinic are in neurological conditions, some traumatic in skull fractures, some in nasopharyngeal tumors, some of inflammatory origin such as cerebro-spinal meningitis, syphilitic basal meningitis, petrositis with Gradenigo syndrome, or in brain tumor cases, such as tumors of cerebellum, midbrain, and the basillar plate. These conditions are all

serious lesions. The traumatic and inflammatory ones often get better under treatment or surgical intervention, and the brain tumor cases, with the exception of the cerebellar tumors, usually die in spite of surgery.

The conditions simulating external rectus paralysis, on the other hand, are seen fairly often, especially powerful internal recti which resemble strabismus fixus as well as external rectus paralysis, and the retraction syndrome or congenital abduction deficiency. Dr. White's differential diagnostic procedures in these conditions, and especially his test for retraction of the globe which is not obvious, are important steps to be taken in diagnosing any particular muscle case that resembles an external rectus paralysis. Dr. White frankly states that a true conception of the status of the ocular muscles may not be possible until the muscles are explored at operation. This I have also found to be true, and I see no reason why the exploration of suspected muscles should not be done. If one hesitates to open the conjunctiva and Tenon's capsule, one may also learn about the resistance the muscle offers to pulling on it by grasping to tendon of the muscle through the intact conjunctiva with a fixation forceps. Then by following Dr. White's "fisherman's judgment" in estimating the character of the muscle and acting accordingly, one will make few mistakes in the muscle surgery of these conditions.

In cases of powerful, overacting interni in young children, occlusion of the fixing eye and abducting exercises for the squinting eye usually help to make the diagnosis by breaking down the convergence spasm, thus ruling out strabismus fixus or external rectus paralysis.

In operating for external rectus paralysis I would follow Dr. White's method. However, I have lately been intrigued by Dr. Wiener's method of cutting off and splitting the insertional tendon of the external rectus and attaching the superior half to the superior rectus and the inferior half to the inferior rectus. This procedure avoids the criticism raised against the operation of transplanting lateral tongues of tendon from the superior and inferior recti to the insertion of the external rectus, which necessarily weakens the action of these two muscles in their respective fields of action. I hope Dr. Wiener is willing to give us the technique of his operation, which I understand he is reporting at the coming meeting of the American College of Surgeons.

I wish to present a case of bilateral congenital deficiency of abduction with retraction, so-called double retraction syndrome, which Dr. White assumes we are all familiar with. Dr. W. G. Mengel of Camden, New Jersey, in reporting a similar case in the June, 1935, Archives of Ophthalmology found 67 bilateral cases in the literature, 33 per cent of which were complicated with facial paralysis.

This 8 year old boy has straight eyes and binocular single vision with perfect fusion in the primary position and in looking straight up or down. The zone of binocular vision is a narrow, vertical area a few degrees to the right and left of the midline beyond which diplopia occurs. As a result, the boy turns his head in looking to either side. All the signs of the syndrome are present, bilateral deficiency of abduction, bilateral deficiency of adduction with marked deficiency of convergence, retraction of the globe when either eye is adducted, narrowing of the palpebral fissure dur-

ing adduction with widening of the palpebral fissure and arching of the brow on the opposite side, oblique movement of the globe when adduction is attempted. No other congenital anomaly is present, the only treatment recommended here was the wearing of glasses for the partial correction of hyperopia of 3 diopters in each eye.

The etiology of double retraction syndrome is probably of central nervous system origin, although the late Dr. Harold Gifford, who read a paper on retraction syndrome at this society in 1924, has recorded good evidence in favor of birth trauma as the cause of the unilateral cases. The unilateral cases are predominantly left-sided corresponding to the predominant obstetrical position L.O.A. Retraction syndrome cases, either unilateral or bilateral, typical or atypical, should be recognized and differentiated from external rectus paralysis, for most of them do not require operation.

Dr. Meyer Wiener, St. Louis: Dr. Gipner requested that I mention that method, which I have not yet reported, of correcting paralytic squint. I wish that we could win over Dr. White to this technic, because in our hands it has been very successful and is giving most satisfactory results.

About 25 or 26 years ago, I performed a Hummelsheimer operation of taking a strip of tendon from the superior and inferior recti and transplanting them to the attachment of the external rectus, and I thought I had an original operation, but when I looked up the literature, I found it had been reported by Hummelsheimer.

This operation was very satisfactory. Sometimes we get five to ten degrees abduction, and at times as much as 35 degrees or more, but every case that I have done has been satisfactory from the standpoint of an improvement.

It is very difficult, I think. You have a small strip of tendon to work with. Lately I have done the modification offered by Dr. Roderic O'Connor of San Francisco, taking a strip of the inner portion of the superior and inferior recti and transplanting them to the attachment of the external recti. In this way we get more pull and more abduction.

If you strip one muscle back further than the other, you are apt to get a slight vertical deviation. Then there is the difficulty of these strips tearing loose from the suture. So I conceived a simpler technic. Instead of mutilating the sound superior and inferior recti and taking a strip and transplanting it to the attachment of the externus, I expose the paralyzed externus and strip it down the center, lift its attachment to the globe, cut it off a-ways back, attach the upper half to the superior rectus tendon, pretty far back, the lower half to the middle of the inferior rectus the same distance back, and we get the same effect with a very much simpler technic and without mutilation of our superior and inferior recti. I have some photographs.

This man was injured in a wreck. He had a brain injury, a complete paralysis of the external rectus with retraction of the interni. I mention this particular case because Dr. O'Connor says those are unfavorable cases. This man was injured, I think, five years before, and had not

FIG. 1.—(Wiener).—Before operation.

FIG. 3.—(Wiener).—Both eyes operated.

FIG. 2.—(Wiener).—Right eye operated.

worked. He is now back at work and driving a car, with binocular single vision. This was done last summer. The operation on the right eye was done by myself at the Missouri Pacific Railroad Hospital, and the operation on the left eye was done by Dr. Vincent Jones, my associate at the same hospital. We have equally good results. I think, possibly, his is a little better than mine.

Figure 1 shows the man as he was, looking straight ahead. He had no power to bring either eye to the middle and had to turn his head considerably to one side in order to see at all. Figure *2* shows the right eye having been operated on, and figure *3* was taken after both eyes had been operated on.

I want to mention one thing more that hasn't been touched upon, and that is a congenital defect we sometimes see where there is a vertical deviation due to paralysis of the inferior rectus and an apparent ptosis. We have had a number of cases, and I have never known just exactly what to do for them. I hit upon an idea this summer. Just recently, on a case where there was complete paralysis in the inferior rectus, complete function of the inferior oblique, complete paralysis of the superior oblique, and incomplete function of the internus and the externus. In this case we couldn't take a strip from the internus and the externus and transplant them below to make the eye pull down, so I took the inferior oblique, which wasn't needed since its opponent was out of function, exposed it with the conjunctival incision, cut it off a distance away from the insertion, and transplanted it a little to the nasal side of the inferior rectus and a little closer to the cornea. It brought the eye down to a level with the right eye, and he is developing ability to pull his eye downward.

Dr. L. C. Peter, Philadelphia: There are several points that I wish to discuss, very briefly, in connection with this very excellent presentation of Dr. White's.

The first is one of nomenclature, which I regard as important. We are confusing three terms or groups of terms in discussing muscles, the question of "ductions," "rotations," "convergence" and "divergence," which are in one group. "Ductions" should apply entirely to the use of two eyes in using prisms, in overcoming the power of prisms. Rotations apply to the movements of one or both eyes in any one of four directions, or oblique directions as well. Convergence and divergence are matters of innervation, something entirely different. I feel we should carefully distinguish between rotations and ductions because if the terms are used discriminatingly, one will always understand exactly what is intended to be conveyed.

The second point I wish to stress is the very excellent suggestion of Dr. White's, that so often we depend upon the correction of a deviation by operating on a single muscle, when the correction should be made by dividing the work between two muscles. Anyone who has operated a great deal knows how much one can increase the correction of a recession operation by employing, at the same time, a moderate degree of shortening of the opposing muscle. As I have stated before, it is something like a game of golf. If one attempts to use a niblick when he should use a midiron, the shot is apt to be weak. The same rule applies in muscle surgery. There are instances when we can employ but one operation, but when one attempts to correct a considerable degree of deviation, it is infinitely better to divide it between two muscles.

The third point I wish to speak of, very briefly, is that of the question of recessions and how far one can recess a muscle without causing any real difficulty in the future use of the eyes. I am referring to the internal rectus. So far as rotations are concerned, a muscle can be resected and reattached to the eyeball as far back as the equator, but when we come to the question of the innervational influence in convergence, you will often find recessed muscles placed back to the equator or farther back are not

adequate to sustain convergence at the nearpoint for any considerable time. The reason for this is quite obvious. Inasmuch as convergence is an innervational condition, the innervation goes into the two muscles equally, both the right and the left, and if the one muscle has been receded too far the convergence will not be very good. In such instances, I think it is much better to divide the work between two internal recti and recede each one an equal degree.

DR. JAMES W. WHITE, closing: I wish to thank the discussors for their partaking in this paper, for their constructive criticism and otherwise.

Dr. Gipner was very kind to me. We are working about the same, and we understand the same language. I thank Dr. Gipner for his trouble in discussing the paper.

Dr. Wiener wishes to convert me. Although I was born in New York State, I am like those from Missouri—I have to be shown. The results I have seen so far from the splicing operations have not convinced me that the same effect might not be obtained if the internal rectus were severed a due amount.

What I mean is this: We say that the external rectus is paralyzed because there is lack of abduction. We don't know whether it is entirely lack of abduction or whether the eye just can't go out because it is held in. Now, to clear up that point, in many of these cases I think we would be perfectly justified, and it would do no harm whatever, in severing the external rectus, as you would do if you were taking any muscle out of the way to do a trephine operation for detachment of the retina. In detachment of the retina, you think nothing of picking up the muscle, ligating it, throwing it off at one side, and when you are through tying it up again, and you have perfect function of that muscle afterward. So in these cases, if we feel the internus has a powerful secondary contracture developed, why not sever that internus and then see if we have any abduction? I think possibly if we did that, the need for the splicing operations would be definitely reduced at least.

There has always been more or less confusion in nomenclature, duction and so on. I think I am not mistaken in saying that I have followed Duane's idea in regard to that—that he thinks the root word "duction" is to carry and abduction means to carry out and adduction to carry in. I may be mistaken. I will look it up.

How far do we recede? That is a question that it is hard to determine. I think it depends entirely upon one's estimate of the strength of the muscle you are going to recede. The first child (picture) I showed had 80 degrees of squint. We are told we can get about 20 degrees recession in one muscle, but I receded only one muscle and got 80 degrees of effect. It would seem that a muscle about three-quarters as strong as that little fellow had would not be receded quite so far and possibly you would get the same effect. I think it is entirely a matter of the field that you get when you operate those, combined with your careful observation of those cases pre-operatively.

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I agree with Dr. Peter perfectly—I think I stated so in my paper, but perhaps not forcibly enough—that in doing any operation on the internus in the way of resection, the nearpoint of convergence must be watched very carefully not to recede either or both of the interni so far that the convergence nearpoint is handicapped. Sometimes you can recede them both to the equator without limiting the convergence nearpoint at all. This little chap that I showed first has a perfectly normal nearpoint of convergence. I receded that internus to the equator. If the nearpoint convergence is poor to begin with, I don't believe the interni should be touched at all, but all the effect got by the resection or what you wish to do to help the interni rectus within a nearpoint of convergence, and I do not operate the interni at all.

CHANGES IN REFRACTION FOLLOWING OPERATION FOR STRABISMUS*

DON MARSHALL, M.S., M.D.

ANN ARBOR, MICH.

IT IS IMPORTANT in orthoptic training and in other methods of correcting all cases of strabismus to obtain the best possible vision for the patient. The only exception is the case in which the eye having better vision is deliberately fogged in some way to stimulate improvement in vision of the amblyopic eye. Except where such a device is purposely applied, every care must be taken to insure the patient's possessing at all times his maximum of visual acuity. To this end, errors of refraction are carefully corrected, and properly fitted glasses become probably the most important single factor in the treatment of strabismus. That glasses often need changing, after operations on the extraocular muscles for strabismus, has received little emphasis by recent writers. In fact, little mention is made in most texts and articles dealing with changes in refraction of the possible effect that surgery may have on the refractive state, and, therefore, on the visual acuity, of eyes which have undergone operative treatment for squint.

A brief outline of a case may show what can occur in this regard. A girl 10 years of age with alternating convergent strabismus of 27 degrees, but with normal vision in each eye, had a recession of the left internal rectus muscle performed, her eyes being thereafter straight with correction. Under atropine cycloplegia her error of refraction eight months before operation had been:

O.D. $+2.00$ sphere combined with $+0.75$ cylinder $\times$ 45 Vision 6/6 - 2
O.S. $+2.00$ sphere combined with $+0.25$ cylinder $\times$ 165 Vision 6/6 - 2

Two days before operation the refraction was:

O.D. $+1.75$ sphere combined with $+1.00$ cylinder $\times$ 60 Vision 6/6
O.S. $+2.00$ sphere combined with $+0.25$ cylinder $\times$ 40 Vision 6/6

* From the Department of Ophthalmic Surgery, University of Michigan. A study completed under the John E. Weeks Scholarship for Research in Ophthalmology, University of Michigan.

These slight changes show that the eyes were not undergoing significant spontaneous change. One week after operation her refractive state, under atropine again, was:

O.D. + 1.75 sphere combined with + 1.00 cylinder × 60 Vision 6/4.5 - 3
O.S. + 0.75 sphere combined with + 2.00 cylinder × 75 Vision 6/6

Four months after operation it had changed to:

O.D. + 2.25 sphere combined with + 0.37 cylinder × 72½ Vision 6/4.5 - 3
O.S. + 2.25 sphere combined with + 0.50 cylinder × 67½ Vision 6/4.5 - 2

Eleven months after operation refraction was:

O.D. + 2.25 sphere combined with + 0.50 cylinder × 70 Vision 6/4.5 - 3
O.S. + 2.00 sphere combined with + 0.75 cylinder × 65 Vision 6/4.5 - 2

If, as Lloyd[1] states, the greatest contribution one can make toward binocular vision is to bring the vision of the poorer eye as nearly to 20/20 as possible, and if the refraction of an eye changes greatly in a week following strabismus operation, it is plain that lenses prescribed for that eye preoperatively may be a gross misfit afterwards, and the patient may be considerably handicapped in his efforts to develop stereopsis.

The original assumption that astigmatism is congenitally acquired and fixed throughout life was early proven wrong. Astigmatism may change at any period of life, but in normal eyes these variations are due to changes in the lens, since corneal astigmatism changes very little at any age.[2 3] Astigmatism may be abnormally caused or changed by corneal section,[4] keratoconus,[4] tumors of the lids as chalazion[5 6] or orbit, trephine operations,[7 8] trauma,[9] tumors or disease of the cornea, pterygium and scars, irregularities in the lens or posterior tunics of the eye, diabetes, and disease of the ciliary body. It has long been felt that traction by the lids and their pressure on the globe cause astigmatism, even to the point of explaining the usual change from so-called direct astigmatism in youth to inverse in old age, due to senile loss of tone of the lids.

In 1874, Noyes[10] reported an increase of astigmatism with rather marked changes in refraction following tenotomy of the internal recti for esophoria. In 1903, Bull[11] reported the disappearance of — 1.75 D. × 90 astigmatism three days after a tenotomy of an external rectus for exophoria. Both of these writers believed the refraction changes were due to the muscle

changes. As a result of these articles, A. E. Davis, in 1903, warned against tenotomy or any surgery as an operative procedure primarily to cure astigmatism. Surgical treatment of astigmatism, chiefly by corneal section or cautery, apparently was having some vogue at that time.[12] Davis stated that most oculists would agree that the rectus muscles exert pressure on the globe, saying "most of us have had the sad experience of seeing vitreous squeezed out of an eye during a cataract operation by traction of the straight muscles on the eyeball, the lids being held entirely free from the eye."[13] Davis referred again to his reported case[14] of a house surgeon at the Manhattan Eye and Ear Hospital who could voluntarily, with lids held away from his eye, increase his corneal astigmatism by 1½ diopters in the right eye, by 1 diopter in the left, and nearly equal this under scopolamine cycloplegic. Without question, this increase in astigmatism, as shown by the keratometer, was caused by the rectus muscles.

That was in 1903. Today, although tenotomy is less frequent, operations for strabismus are becoming more and more frequent, while the effect of these on errors of refraction seems to be almost forgotten. Perhaps this reticence is to avoid an implied connection with the school of the late Dr. Bates,[15] who developed this subject to a ridiculous extreme. Ball[16] and Duke-Elder[17] mention that tenotomy of a rectus muscle may alter the corneal curvature. The American Encyclopedia of Ophthalmology mentions it, the section having been written by Davis.[18] Posey,[19] Jackson,[2 4 20] Clark,[21] Ellett,[22] and Fuchs[23] do not mention it. But twenty years ago Howe[24] was convinced that the extraocular muscles affect the form of the globe, and showed incidentally that the internal rectus muscle has a lifting power of 14 gms.; and thirty-six years ago Norris and Oliver specified "contraction of the exterior ocular muscle groupings" as a cause of astigmatism.[25]

PROCEDURE

To discover and establish the occurrence of changes in refraction consequent upon muscle operations in strabismus, a series of cases was studied. The group consisted of 55 patients who were operated upon on 62 occasions to correct their squint, but who showed a total of 88 muscles operated upon, since often two or more muscles were dealt with at one sitting. The group was selected only by excluding all cases with poor vision or poor fixation in either eye, and all patients who were unreliable at

refraction. No selection was made as to sex, age, type or degree of squint, or type of operation. These patients were refracted under cycloplegia before operation, again on discharge from the hospital, and in a large share of the cases again at later intervals. The cycloplegic consisted of 1% atropine sulphate solution instilled three times daily for three days in children of 14 years of age or under, and a combination of 3.5% solution of homatropine hydrobromide and 0.5% cocaine hydrochloride instilled every ten minutes for six doses in patients over 14. A large majority of the refractions were performed by the same examiner. All patients were retinoscoped and then checked subjectively.

RESULTS

The age of the 55 patients studied ranged from 5 to 31 years, averaging 12.3 years. Types of squint represented were: Convergent 83.5%, 43.6% being alternating; Divergent 14.5%; Right Hypertropia 1.8%. The right eye was operated upon alone in 38.7%, the left eye in 48.4%, and both eyes in 12.9%. In nine, cases in the right eye, and also in the left, two muscles were operated on at the same sitting. Operations performed were as follows: Recession 50%; Resection 35%; Tenotomy 8%; Tuck 5%; Resection with Advancement 2%. In 40% of the 62 operations the patient had been refracted at some time at least several months previous to the operation, which thus provided a check on any marked changes in their refraction which might be occurring spontaneously. The time of this early refraction averaged 19½ months previous to the operation. In slightly over half of these cases the difference between this early refraction and that done immediately before operation did not exceed 0.25 diopter in either vertical or horizontal meridians. Of the remaining eyes, 45% showed a change in one meridian of only 0.25 D., and in 23% a change of only 0.50 D. The remaining small portion showed greater changes, but this early refraction indicates that, in general, the eyes in this study group were not undergoing any marked spontaneous change in their refractive state.

Seventy-seven per cent of the patients had their basic refraction, from which subsequent changes were calculated, determined four days or less before the day of operation, and 94% less than three months. The amount of squint preoperatively averaged 25 degrees. Ninety-four per cent of the patients had their first postoperative refraction fourteen days or less after

the day of operation. Average vision of all eyes was better than 6/6.

Changes in the refractive state are reported here as changes in the vertical and horizontal meridian of the eye, considering the latter as a refracting unit. As two examples, an increase in convex sphere discovered at the time of refraction is here noted as a decrease in the curvature of both the vertical and horizontal meridian of the globe, of an equal degree in each plane. A decrease in the strength of a convex cylinder, axis vertical, required by the eye, signifies an increase in the refracting power of the eye in its horizontal plane, and is here so noted. In oblique axes, the listing is with that division to which the axis is nearest.

Although the refractions were done with great care, and every effort was made to have each examination accurate to one-quarter of a diopter or less, all changes of less than 0.50 D. are here ignored. Changes considered significant in this study are all 0.50 D. or greater.

First Postoperative Refraction

Of the 70 operated eyes in 62 patients, 60% showed a change of 0.50 D. or more in one or both meridians. Of the 54 untouched eyes in this group, 41% showed such a change. In 20% the change was greater in the unoperated than in the operated eye.

Of the 42 cases showing a change of 0.50 D. or more in the operated eye, the average change in the vertical meridian was 0.86 D. In the horizontal plane of the eye, the average change was 0.65 D.

Subdividing the group according to the type of operation, and excluding all changes of 0.25 D. or less, we find, when only a lengthening operation (recession or tenotomy) was performed on one or both eyes (23 patients or 25 eyes), 60% of the eyes showed significant change in the vertical meridian of the globe, of which 93% were an increase in curvature averaging 0.79 D. In the horizontal plane, 32% showed significant change, of which 87% were a decrease in curvature, averaging 0.57 D.

When only a shortening operation (resection, advancement or tuck) was performed, and no simultaneous lengthening operation (16 cases, 17 eyes), 35% showed a change of over 0.25 D. in one or both meridians. Such a change in the vertical meridian was found in 24%, and in the horizontal plane 24%. The vertical curvature was found to be decreased three times averaging 0.83

D., and increased once 0.50 D. The horizontal curvature was increased three times averaging 0.67 D., and decreased once 0.50 D. Thus 75% of the significant vertical changes consisted of a decrease in refraction power, and 75% of the horizontal changes an increase.

When both a lengthening and shortening operation was done on one eye (17 cases), 77% showed significant changes in the vertical meridian, of which 77% were an increase in curve, averaging 1.05 D., and 23% a decrease averaging 0.83 D. In the horizontal plane 41% changed, all a decrease averaging 0.75 D.

In the first, or lengthening operation group, 68% of the unoperated eyes showed no significant change in either meridian; 18% changed in the vertical and 27% in the horizontal meridians. Of the 15 unoperated eyes in the second, or shortening operation group, 40% showed significant change, always a flattening, and usually of 0.50 D. In the third or combined group, the unoperated eyes showed change in the vertical plane in 29% and in the horizontal in 12%. Except for 25% of significant changes in the vertical meridian in the first and third groups, all changes in the unoperated eye were decreases in curvature.

FIRST POSTOPERATIVE REFRACTION — OPERATED EYE

TYPE OF OPERATION	M	SIGNIF. CHANGE	TYPE	AVERAGE OF THESE
Length.	V	60 per cent	93 per cent increase	0.79 D.
	H	32 per cent	87 per cent decrease	0.57 D.
Short.	V	24 per cent	75 per cent decrease	0.83 D.
	H	24 per cent	75 per cent increase	0.67 D.
L. & S.	V	77 per cent	77 per cent increase	1.05 D.
	H	41 per cent	100 per cent decrease	0.75 D.

TABLE 1 — A summary of the changes in refraction of 0.50 D. or more occurring between the examination made immediately before operation and that done at the time of discharge from the hospital. M = Meridian; V = Vertical meridian of the globe; H = Horizontal; L. & S. = Operation where both a lengthening and a shortening operation were performed on the same eye. Increase = Increase in curvature, or refracting power; Decrease = Decrease; D. = diopter.

A study of the relation between the amount of refraction change and the amount of squint change, both resulting from the surgery performed, shows the average squint change at the time of the first postoperative refraction to have been 19 degrees. Dividing all cases into two groups, we find that of 62 operations, 27 showed a change in the amount of squint of 19 degrees or

less, or slightly less than half the cases, the other half showing a change of 20 degrees or more. Of the 42 operated eyes showing a postoperative change of 0.50 D. or more, 11 were in cases of the first half, and 31 in the second. The amount of total change in refraction in the two halves was exactly the same, but the frequency of such changes was nearly three times as great (11 to 31) when the squint was changed 20 degrees or more by surgery. Similarly, in the 22 unoperated eyes that showed significant change, 9 were in the first half, of changes under 20 degrees, and 13 over 20 degrees. Again the amount of change was identical in the two groups, but the frequency of change was 44% greater in the latter.

Second Postoperative Refraction

A subsequent refraction was made on 36, or over half of the patients, at a period averaging six months after the operation. Of the 38 operated eyes, 74% showed significant change from the first postoperative refraction in one or both meridians, and 62% showed such a difference from the preoperative refraction. Thus, three-quarters of the eyes were continuing to change, and two-thirds were still definitely different from their state before operation.

In the unoperated eye, 41% showed change from the last refraction, and 32% from the preoperative refraction. With one exception these were only in one meridian of the eye and only 0.50 D. Changes in the vertical meridian were nearly three times as frequent (4 to 11) as in the horizontal, but there was an even division between increases and decreases in curvature.

Between the first and second postoperative refractions, changes in the operated eye were 33% more frequent in the vertical than in the horizontal meridians. In the vertical meridian, 63% showed significant change. Of these, 87.5% were changes in the direction back toward the preoperative state and only 12.5% a continuation onward of a difference first noted at the first postoperative refraction. In the horizontal meridian 72% of the significant changes were back and only 28% onward. In the unoperated eyes (34) only 32% showed change in the vertical, of which over half were back, but nearly as many onward; and in the horizontal only 12% showed change, equally divided between the two. In the operated eye, 81% of the significant changes were back; in the unoperated, 53%.

A comparison of the total amount of refraction change was made by adding the change in the horizontal and vertical meridians, after excluding all changes of 0.25 D. or less. By comparing these total changes we find that between the preoperative and first postoperative refraction, and between the first and second postoperative refractions, the following were the average changes:

OPERATED EYES

	1ST POSTOP.	1ST TO 2ND POSTOP.
Lengthening Operation	0.59 D.	0.65 D.
Shortening Operation	0.34 D.	0.46 D.
Lengthening and Shortening	1.09 D.	0.95 D.

UNOPERATED EYES

	1ST POSTOP.	1ST TO 2ND POSTOP.
Lengthening Operation	0.25 D.	0.07 D.
Shortening Operation	0.30 D.	0.30 D.
Lengthening and Shortening	0.29 D.	0.35 D.

There was a slight tendency for changes present at the second postoperative refraction to be greater when the amount of squint change was great, in both the operated and unoperated eyes.

With a definite tendency manifest for changes to disappear as time goes on, it is of interest to note the changes existing at the second postoperative refraction from the preoperative state, or an analysis of the semipermanent changes in the eyes.

In the group in which the lengthening operation was done, there were 17 operated and 14 unoperated eyes. Of the operated eyes, 59% showed a significant change in one or both meridians. In 75% the vertical change had been an increase in curvature, and in 87% the horizontal a decrease. In the unoperated eye, 10 of 14 showed no significant change. All changes noted were decreases of curvature.

In the shortening operation (5 cases) 40% showed significant change in the operated eye. All changes were decreases of curvature. In the unoperated eye only one of five showed change.

In cases where both a lengthening and shortening operation had been done on the same eye (16 operated and 15 unoperated eyes), in the operated eye 69% (11) showed significant change in one or both meridians. In the vertical curvature, 75% were increases in curvature. In the horizontal curvature, 86% were decreases. In the unoperated eyes, in 60% no significant changes occurred in either meridian. Vertical and horizontal changes were of the same type, on the average, as in the operated eyes.

Third Postoperative Refraction

At an average time interval of 11.6 months after the operation, ten cases were refracted still a third time. One only of these (a shortening operation) showed no significant change in either meridian of the operated eye. Six of the ten unoperated eyes showed no change. Differences between the preoperative and this third postoperative refraction were similar to the changes found at the second postoperative refraction. Changes were noted as follows:

Lengthening Operation (4 cases):

Operated Eye—In the vertical meridian, 3 out of 4 showed change, each an increase in curvature, averaging 0.83 D. In the horizontal meridian 3 out of 4 showed change, two a decrease averaging 0.75 D., and one an increase of 0.75 D.

Unoperated Eye—Two cases showed significant change.

Shortening Operation (3 cases):

Operated Eye—Two eyes showed change, one a decrease of 0.50 D. in the vertical plane, and one a decrease of 0.50 D. in the horizontal.

Unoperated Eye—One showed an increased curve in both planes.

Lengthening and Shortening Operation (3 cases):

Operated Eye—One showed an increase in vertical curve of 0.50 D. The other two showed decrease in the horizontal plane averaging 0.82 D.

Unoperated Eye—One showed an increase in the vertical curve of 0.75 D.

Of these three groups, those who had undergone a simple lengthening operation showed the largest and most frequent changes. Among these ten cases, the average change in amount of squint was 19 degrees at the time of examination.

CHANGES IN REFRACTION

Percentage of Significant Change

EYE	0 TO 1	0 TO 2	1 TO 2	0 TO 3
Operated	60 per cent	62 per cent	74 per cent	90 per cent
Unoperated	41 per cent	31 per cent	41 per cent	40 per cent

Key to Refractions:

0 = Preoperative
1 = 1st Postoperative
2 = 2nd Postoperative
3 = 3rd Postoperative

TABLE 2—A summary of significant changes in refraction in the total operated and unoperated eyes occurring between the refractions as indicated.

CONTROL GROUP

All the changes discussed above might be ascribed to inaccurate refractions. To determine the consistency of our refractions, a control group of 19 unselected young (12 through 21 years, inclusive) patients from our tuberculosis unit were refracted twice. None was refracted at an interval of less than two weeks. Seventeen of the 19 were done two or three weeks apart. The other two were done at intervals of 11 and 30 weeks, respectively. Cycloplegic and technique were as in the main group.

The average age of this control group was 18 years. Of the 38 eyes, 33 (87%) showed no change over 0.25 D. in any meridian. Two eyes changed 0.37 D. in one meridian, less in the other, and in three eyes there was a change of 0.50 D., but in one of the latter the two refractions were seven months apart. It seems fair to conclude that, taken as a whole, the refractions done in this study are sufficiently accurate to warrant the drawing of conclusions from them.

Although closely related to the problem of this study, the academic question of how such changes in refraction occur as a result of surgery on the extraocular muscles, and in what portion of the globe they are manifest, cannot be adequately treated here. Theory and correlative evidence would point to the cornea as the structure which by a change of curvature would cause these changes in refraction in various meridians. It is unlikely that the effect could result from a change in the ciliary muscle, lens, or posterior portion of the eye. Those authors who mention the effect of the extraocular muscles on astigmatism unanimously agree that their action is on the cornea alone. To prove this point, keratometer readings were taken on a portion of the cases in our study group. For factors not easily controlled, the entire group could not be so examined, and the relatively few measurements made would not warrant the drawing of conclusions from them. This aspect certainly demands further study, but from a clinical and practical viewpoint, the occurrence of changes in the total refractive error is the significant fact, whether they be actually limited to the cornea, as is most likely, or not.

Similarly, the mechanism of refraction changes in one eye following operation upon the muscles of the other eye is of considerable interest. Superficially, it would seem that gross errors had been made in the examinations. But such changes appear not illogical in a consideration of the conjugate functioning of the

extraocular muscles of the two eyes. Jackson[26] states "an operation on any one of the muscles, if it alter the effect produced by contraction of that muscle, alters the general muscular balance." Since the two eyes by innervation function together in conjugate movements and in convergence, it follows that surgical change of tension of one or both of the horizontal rectus muscles of one eye might by reflex compensation alter the tension of the horizontal muscles of the opposite eye. Such changes appear to be relatively infrequent, minor, and without pattern, in comparison with the operated eye, yet frequent enough to warrant as careful observation as those in the eye on which surgery was performed.

CONCLUSIONS

Reviewing the results, we may make the following general conclusions:

1. The refractive state of an eye usually changes as a result of operation upon the extraocular muscles of that eye. These changes are more frequent when a lengthening operation is done, with or without a simultaneous shortening operation, and when a large deviation is corrected. Sixty per cent showed a change of 0.50 D. or more in at least one meridian when discharged from the hospital postoperatively. The average change is three-quarters of a diopter among the 60% noted.

2. The opposite untouched eye may show changes, though less frequently; 41% of the unoperated eyes showed significant change when discharged postoperatively.

3. Later observations show that in the great majority of cases the eye tends to return to its preoperative state slowly, but since this tendency is often incomplete or overdone, permanent differences usually result. Six months postoperatively 61% of cases showed significant change from the preoperative state in the operated eye. A year after operation nine out of ten cases showed such a difference. It cannot, therefore, be assumed that changes due to surgery will in time correct themselves and disappear, even though they do show such a trend.

4. Refraction changes from lengthening and from shortening muscle operations correspond to the effect the increase or decrease of muscle tension consequent upon the surgery would have upon the globe, if such changes are in the corneal curvature. Thus, the usual change following a recession operation is an in-

crease of vertical refractive power and a decrease of refractive power in the horizontal meridian. Following resection of a lateral rectus, the opposite usually occurs.

5. Although the type of change may be anticipated, in general, developments in the individual case are so uncertain in type and degree that the final result cannot be foretold.

6. The frequency of changes in both the operated and unoperated eyes points to the necessity of frequent postoperative check of visual acuity, with refraction and change of lens whenever indicated.

SUMMARY

1. Recent literature makes little note of the changes in refraction caused by surgery on the extraocular muscles.

2. Pre- and postoperative examination of a series of patients on whom surgery was performed to correct strabismus showed significant changes in both the operated and unoperated eyes.

3. These facts show the importance of frequent check of vision and refraction after such operations, if postoperative orthoptic training is to be adequately performed, and the best possible vision provided for the patient.

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DISCUSSION

DR. J. L. BRESSLER, Chicago: Dr. Marshall calls to our attention the possible occurrence of changes of refraction in the eyes following surgery for strabismus. Many of us have considered this possibility before and have dismissed it as negligible.

If these changes are substantiated by further investigations of Dr. Marshall and others, we will indeed be indebted to the essayist for an important contribution to ophthalmology.

In my attempt to check the findings of Dr. Marshall, I accumulated a series of thirteen cases. This is a small group compared with Dr. Marshall's fifty-five cases, but I received a description of his method and routine too late to follow more of my own cases in a like manner. I will try to arrange my findings in such a way that they may be easily compared with Dr. Marshall's report.

Of these thirteen cases, eight were alternating convergent, two were alternating divergent, two were right monocular convergent, and one left monocular convergent. The angle corrected averaged 23.8 degrees. Of these, nine were corrected more than 20 degrees, and four less than 20 degrees. The types of operation performed were as follows:

1. Ten cases had both a recession and a resection on the same eye.
2. One case had a resection only.
3. One case had a resection with advancement and a tenotomy.
4. One case had a tucking and a tenotomy.

No case in my series was operated more than once.

A résumé of the changes of the refraction in this series shows the following:

1. In six cases, no change greater than 0.50 D. occurred in either meridian of the operated or unoperated eye.
2. In six cases, changes in the operated eye varied from 0.50 D. to 2.25 D.
3. In one case no change occurred in the operated eye, but the unoperated eye showed an increase in the vertical meridian of 1.25 D. and in the horizontal meridian a decrease of 1.25 D.
4. Of the unoperated eyes, other than the one just quoted, only two showed changes greater than 0.50 D. in either or both meridians.
5. Only three of the operated eyes in this series showed changes enough to be concerned about. They varied from 1.00 D. to 2.25 D. either in one or both meridians.

In contrast to Dr. Marshall's findings — his cases showed the greatest refractive changes in the eyes that had the greatest correction of the angle of deviation — several of my cases with angle corrections varying from 29 to 33 degrees showed changes of less than 0.50 D. Only one case, in which the correction was 31 degrees, showed a flattening of the horizontal meridian amounting to 2.25 D. The few cases that did show changes greater than 0.50 D. occurred more frequently in the lower bracket of corrections. All of my patients had refractions two to four days preoperatively and again, ten to fourteen days postoperatively. Some also had a third refraction one month later.

One patient of this series has been under observation for several years. She was operated upon one year ago. I did a preoperative refraction, but no postoperative refraction until now, one year later. This showed an increase of 0.25 D. in both meridians of the operated eye, and a reduction of 0.25 D. in the unoperated eye. These changes, in my estimation, are certainly not great enough to be concerned about.

This series of cases does not show the marked changes described by Dr. Marshall. That some changes do occur in a large number of patients after operation for strabismus is probably true. Whether or not the changes are great enough to be significant, we must determine by further investigation. I feel this series of cases is too small on which to base a final opinion. If, as Dr. Marshall found, most of the eyes return to their previous refraction, the occurrence of changes in refraction becomes a less alarming fact. I would suggest, as did Dr. Marshall, that instead of doing refractions before surgery, this should be done one month or more following the operation.

Another factor influencing the changes described by Dr. Marshall may, in my opinion, be the type of surgery performed. A lengthening or a shortening operation alone may produce a change in the tension on the cornea, but if one does a carefully-balanced recession and resection on the same eye, very little change will occur. In this series of thirteen cases, ten had a recession and resection on the same eye. Six of these showed practically no refractive changes. This leads me to believe that careful surgery will go a long way in eliminating marked changes in refraction.

In conclusion, I wish to say that, inasmuch as some changes in the refractive state of the eyes do occur following surgery for strabismus, these changes, small or large, should not be ignored. Refractions should be done postoperatively and checked frequently. Further investigation of this subject should be followed and reports made.

Dr. Oscar Wilkinson, Washington, D. C.: I am sure we all thank Dr. Marshall for calling our attention to this change in refraction, but I must say that from our observations of the past twenty years I have seen very little change in refraction in squint cases, and I feel that these changes can be accounted for largely by the type and method of operating.

I noticed in the first case reported that he said he corrected something like a 25 or 30 degree deviation with one recession. I feel sure that anyone who corrects a 25 degree deviation with one recession is doing too much recessing. In other words, you have deprived that eye of the normal amount of support that that internal rectus should be to that eyeball.

I have maintained for years that Dr. Curdy of Kansas City should have more credit for what he taught us in 1916. His idea was to operate on both muscles in order that you might balance the amount of correction that you are giving. Any eye that has a recession as far back as the meridian, as far back as Dr. White suggested in his previous paper, has lost the entire support of that muscle, and I don't believe we are justified in doing too much recession or too much resection. Operate on the other eye, if necessary, but don't do too much recession. The recession operation in cases in which you hope to get after binocular vision will certainly interfere with the ability of that patient to do work, because the convergent power will always be weaker than it should be.

DR. DON MARSHALL, closing: I wish to thank Dr. Bressler for his remarks and for carrying out, as far as he was able in the short time available, a similar series of studies on a similar group of cases. I do agree with him absolutely in his conclusions. In the past year and a half, I have discussed this matter with several men and general skepticism has been brought forth about the significant or large change in refraction occurring after squint operations. As far as the percentage of such changes which do occur is concerned, obviously the exact figure must be determined on a very large series of cases and on the basis of examinations by many men, not on one series of cases studied by one man.

I do feel, however, that the series presented tends to show that changes do occur, perhaps not in each case and perhaps usually so slight as to be almost insignificant, but now and then a case does come up in which there is a relatively large change in refraction, so large that if that patient were to continue with the glasses provided for him on the basis of preoperative test he would have very much diminished visual acuity in the eye which shows the change.

In reply to Dr. Wilkinson, regarding the amount of recession done on the illustrative case of which you saw a slide, the recession was only 5 mm. With no exception do we ever do a recession further back than that. The young girl did show a correction of 25 degrees, measured by the Priestley-Smith method with her glasses. Her eyes, after the operation, were straight with the correction. Without her glasses, there was considerable convergence. In all our surgery for strabismus we follow exactly the same principles laid down by Drs. P. C. Jameson and James W. White.

I feel that enough changes do occur and changes large enough occur in the refraction of patients who have had a squint operation to warrant careful observation by those who have such patients under their care and to justify further study along these lines.

STUDIES ON THE INFECTIVITY OF TRACHOMA*

L. A. Julianelle, Ph.D., and
R. W. Harrison, Ph.D.
BY INVITATION
ST. LOUIS

When the study of trachoma was instituted in 1930 at Washington University under a grant from the Commonwealth Fund of New York, it was decided that the most profitable results would be attained by temporarily disregarding the bulk of accumulated literature on the etiology of trachoma and by undertaking a study in the manner that is usually employed with a new disease. The reason for this decision was primarily the conflicting and numerous opinions concerning the causation of trachoma. It has been necessary, therefore, to repeat a number of experiments performed by previous investigators in order to establish fundamental conditions from which future work might be projected.

Earlier communications from this laboratory have already contributed corroborative evidence that trachoma is transmissible to animals and that it is infectious in nature. That the experimentally induced infection is specific and unrelated to deficient or faulty nutrition either as an associated or complicating factor has also been recorded. As others had shown previously, further experiments indicated that under specified conditions, the infectious agent of trachoma does not readily traverse Berkefeld filters. Concurrently with these studies, an investigation conducted on the bacteria cultivable from trachoma and clinically similar conditions revealed that trachoma is not characterized by a particular bacterial flora, and that the bacteria isolated from trachoma may be encountered with similar frequency and distribution from folliculosis, chronic conjunctivitides of unknown etiology, inclusion blennorrhea and, to some extent, even from normal eyes. Repeated attempts to induce specific infection with bacteria cultivated from trachoma, either individually or pooled in the proportions occurring on the conjunctiva, made inevitable the conclusion that bacteria grown from trachomatous material, itself

* From the Oscar Johnson Institute, School of Medicine, Washington University, Saint Louis. Conducted under a grant from the Commonwealth Fund of New York.

active in inducing experimental trachoma in monkeys, are incapable of provoking the conjunctival reaction regarded as characteristic of experimental trachoma. The organisms isolated were predominantly diphtheroids and staphylococci; the three varieties of Streptococci as determined by their activity on blood; Pneumococcus, usually group IV; a number of gram positive rods including chromogens and Morax-Axenfeld bacillus; gram negative cocci, but not gonococcus; a large number of gram negative rods including Koch-Weeks bacillus and Bact. granulosis; yeasts and filamentous forms.

Further experiments were undertaken to obtain information on the epithelial cell inclusion first described by Prowazek and Halberstadter. The data indicate that trachomatous tissues containing inclusions are frequently non-infectious for monkeys, and conversely that trachomatous tissues lacking inclusions are frequently infectious. Monkeys that failed to react to injections of materials containing inclusions were subsequently demonstrated to be susceptible to infection, thus indicating that lack of infection on the first occasion was not due to a high resistance of the animal, but actually to an inability of the inclusions present in the first material to cause experimental trachoma. Moreover, repeated examinations of inoculated monkeys, both infected and non-infected, regularly failed to reveal the presence of inclusion bodies. In a number of monkeys, examinations were made daily during the period of incubation, but again inclusions were never observed. It is impossible at the present time to draw any conclusions from these experiments. Whether this indicates that inclusions are found only in the human in whose tissues the agent of trachoma is highly parasitized, or that the inclusions are only concomitantly and not causally present in trachoma is not revealed by the foregoing experiments.

Since it was found that (1) Berkefeld filtrates from infective tissues are usually non-infectious, (2) bacteria cultivated from infective tissues are likewise non-infectious, and (3) inclusions do not necessarily accompany infectivity at a given time, it was proposed to continue the studies by investigating the possibility that the incitant of trachoma might be a virus but non-filterable. Whether it be non-filterable because it is contained within epithelial cells which are themselves unable to penetrate filters or whether it be non-filterable in the sense that the virus of smallpox is non-filterable, did not appear to be of moment for the time being. So also it did not matter if temporarily the

inclusion be disregarded, because if causally related to trachoma it would necessarily be encountered in the anticipated experiments.

A study was made of cultivating the agent of trachoma in growing tissue cultures. Cultivation was attempted in minced chick embryo according to the technique devised by Rivers, in the developing egg (Goodpasture method), and in various tissue blocks in plasma (Carrel method), and incubation was carried out at both 37° C. and 30° C. The technical difficulties were numerous, the most consistent being the presence of adventitious bacteria in the eye. They were almost always present in the seedings from the trachomatous conjunctiva and they grew rapidly in the tissue cultures with the result that the tissue culture cells were suppressed or killed. Numerous methods and chemicals were employed hoping that selectively the agent of trachoma might survive while all the extraneous bacteria might be destroyed. It was found, however, that the agent of trachoma is less resistant to injurious agents than its accompanying bacteria. Eventually sufficient tissues were obtained free of cultivable bacteria. In these instances smears and sections of the growing tissue cultures disclosed no visible organisms or inclusion-like structures. A number of the tissue cultures were ground and inoculated into susceptible animals. Some of the cultures contained bacteria, while others were bacteriologically sterile. In no instance, however, was it possible to infect monkeys with the tissue cultures. These experiments are still in progress, so that it is not desirable to offer, at the present time, any conclusions on the cultivability of the agent of trachoma.

When the tissue culture experiments were well under way, an attempt was made to demonstrate the agent of trachoma by testicular passage. The technique employed consisted of collecting trachomatous material obtained by grattage in 1.5 cc. of broth per patient. The material from two patients was pooled and then (1) seeded into tissue cultures and bacteriological media, (2) inoculated in monkeys, and (3) 0.5 cc. was injected in each left testicle of two rabbits and two guinea-pigs. No significant information was gained from the tissue and bacteriological cultures. The original material was found to be of a low grade activity. While monkeys were infected, the infection subsided within a month. Material obtained from one rabbit and one guinea-pig, one week following injection by removing and emulsifying the inoculated testicle, showed no increased infective capacities. Testicular material of the second animals two weeks after inoculation was found to be highly infectious when injected

in the conjunctiva of monkeys. The incubation period was shortened to five days and the reaction of folliculosis, etc., became rapidly intense. The infectious testicular material was passed through a second animal testicle with a distinct diminution in infective power; after a third passage, the infectious agent was either completely lost or completely attenuated. On the other hand, alternate passage from rabbit testicle to monkey to rabbit, etc., has been continued now for several passages.

Examinations for organisms were made by smear preparation, bacteriological culture, and tissue culture of the original human material, the infected monkey conjunctiva and the rabbit and guinea-pig testicles. In addition, tissue sections were also examined in the case of the experimental animals. The human and monkey tissues showed (1) no significant bacteria, (2) no inclusions, and (3) no growth of the infectious agent in tissue cultures. The testicles were examined with great care, but no sign of any organism, whether bacterial, inclusion, virus, or other variety, was demonstrable. Elaborate tissue cultures of the infected testicles yielded cultures free of bacteria, and incapable of infecting monkeys.

For purposes of control, material obtained in the same way from six patients with folliculosis was studied in a similar manner, but in no instance was it possible to demonstrate an infectious agent. Material obtained from two patients with a chronic infectious conjunctivitis of unknown etiology was also studied in parallel experiments without inducing any clinical signs in monkeys. In another experiment, material obtained by grattage from a patient with inclusion blennorrhea and showing numerous epithelial inclusions also failed to demonstrate the trachomatous agent after testicular passage. A final control experiment was performed with hog-cholera virus which was first proved capable of inducing fatal infection in hogs. When inoculated directly in monkeys, or following two weeks' passage in rabbit testicle, it failed to produce any significant reaction.

Since the first experiment which has been reported in detail above, nine more experiments have been conducted on testicular passage of the agent of trachoma. In eight, the original and testicular passage material was found to be non-infectious as judged by inoculation of monkeys. The remaining material, after testicular passage, was found capable of inducing experimental trachoma. Here again the tissue cultures were consistently sterile in each instance, and careful examination of the inoculated testicles gave no evidence of any organismal forms.

To summarize, then, of ten complete experiments in which trachomatous material was studied by testicular passage, two were originally infectious for monkeys. After testicular passage, the same two materials were infectious in greater degree than originally, while the eight specimens non-infectious in the beginning, remained non-infectious after testicular passage. It seems, therefore, that unless the trachomatous agent is present originally, testicular passage does not allow recovery of the agent; when the trachomatous agent is present in the human tissue it is recoverable by testicular passage in greater potency and in a purified condition.

Except for a minor degree of inflammatory reaction, the tissue changes in the testicle are insignificant. The animal exhibits no general reaction, and it is apparently difficult to maintain the activity of the trachomatous agent by continuous testicular passage. A few preliminary experiments in filtration of the infected testicular tissues both by Berkefeld and Elford filters have thus far been unsatisfactory and inconclusive. So also concentration of the trachomatous agent by cataphoresis of the infected testicle has not added any information on the nature of the agent. Both filtration and cataphoresis of testicular tissues are still under study.

It is not desirable at the present time to draw any conclusions based on the testicular experiments. While the indications are that given an active trachomatous material, the infectious agent may be purified and perhaps increased by testicular passage, a great deal more study is required to determine the validity of such a conclusion. It is, therefore, not possible to interpret the significance of these experiments at the present time. The studies are being reported not to define the infectious agent of trachoma but merely to record the adoption of a method new to the study of the etiology of this disease. Whether this method will prove fruitful or not must await future experiments.

DISCUSSION

DR. PHILLIPS THYGESON, Iowa City, Ia.: Doctors Julianelle and Harrison have summarized the results of their important trachoma investigations of the past two years. They have confirmed the transmissibility to monkeys and specific nature of the disease and have established beyond doubt that trachoma is unrelated to vitamin deficiency or other nutritional defect. Their careful studies of the bacterial flora in trachoma have indicated that the disease is caused neither by repeated infection with ordinary conjunctivitis-producing bacteria, as has been claimed by Williams and her associates from the laboratories of the New York City Department of Health, nor by a specific bacterium such as Bacterium granulosis as has been claimed by Noguchi and by Olitsky of the Rockefeller Institute.

The great majority of the workers in this field will now agree that the agent of trachoma is not a bacterium.

By a process of elimination, the essayists have reached the tentative conclusion that the agent of trachoma is invisible, non-cultivable by ordinary methods, and non-filterable through Berkefeld filters. That the agent of trachoma belongs to the group of so-called viruses many workers will agree, but that the agent is necessarily invisible will certainly be contested by some. Failure of many investigators to produce active filtrates with Berkefeld filters suggests that the particle size of trachoma virus is, like vaccinia virus and other viruses which filter with difficulty, relatively large. Viruses of small particle size, as for example the virus of foot and mouth disease, generally filter with ease. There is now known to exist a group of viruses, typical examples of which are vaccinia virus and psittacosis virus, whose particle size, as determined by centrifugal analysis and by filtration through collodion membranes of graded pore size, lies between 0.1 and 0.2 microns. These viruses lie just at the limits of the resolvability of the microscope, but by appropriate staining methods can be seen as minute coccus-like structures, known as elementary bodies. (For a recent review of the status of the elementary bodies in virus diseases consult J. C. G. Ledingham, Bull. Johns Hopkins Hospital, 57:32, 1935). The presence in trachoma of such minute bodies, the elementary bodies of Halbersteadter and Prowazek, necessitates their consideration as possible causal agents of the disease.

Doctors Julianelle and Harrison have considered this phase of the problem in terms of the epithelial cell inclusions rather than in terms of the individual elementary bodies which form the inclusions. They have concluded that materials not containing inclusions may be infectious for monkeys, but since they did exclude free elementary bodies from consideration their conclusions are open to criticism. Recent studies at the University of Iowa indicate that the inclusions from which the elementary bodies are necessarily derived can be found by persistent search in all cases of trachoma whose activity is of such order as to be useful in transmission experiments. It is, therefore, our belief that in direct transmission experiments the possibility of transfer of free elementary bodies cannot be excluded.

More serious evidence against an etiologic rôle for the elementary body is contained in their findings that M. rhesus monkeys experimentally infected with trachoma do not show inclusions. In this connection, however, it must be remembered that the Halbersteadter-Prowazek inclusion was first seen in experimental trachoma of the ape and that Lindner and other reliable observers have reported inclusions in experimental trachoma in the baboon. In these animals a disease of considerable intensity occurs, while in the M. rhesus, which has been employed by the St. Louis workers, only a localized follicle formation results. We have noted that in human cases the number of demonstrable inclusion bodies is about in proportion to the severity of the disease and that in cases with acute or subacute onset, or with acute or subacute exacerbations during the course of the disease, the inclusions may be numerous. On the other hand, in very mild cases, the inclusions may be demonstrated only by prolonged search. Since the intensity of trachoma in the experimentally

infected rhesus monkey is well below that of even the mildest of human cases it may well be that the inclusions are present in numbers below the limits of bacterioscopy. That this is probably true is indicated from the study of inclusion blennorrhea in which the experimentally infected rhesus monkey may show inclusions in demonstrable numbers only during the incubation period, whereas the much more susceptible baboon often shows numerous inclusions. In addition, two experimental transmissions to human eyes have been accompanied by readily demonstrable inclusions.

By their use of testicular passages, the St. Louis workers have opened up a new field of attack on the trachoma problem. It is to be hoped that their future studies will determine whether actual multiplication of the virus in the testicle occurs or whether the virus is simply maintained in an active state without increase.

Dr. M. M. Cullom, Nashville, Tenn.: In regard to the manner in which trachoma is acquired by the patient, I wish to set down the conclusion I have arrived at as a result of 38 years of experience. There are many who claim that the disease arises as a result of malnutrition and that it is not a true infection. A distinguished member of this Society, the late Dr. J. A. Stucky, wrote:

> "I have never seen a case in which there was unmistakable evidence that it was contracted by contact with one having trachoma. I believe genuine trachoma will be proven to be a deficiency disease, a term sanctioned by custom and embracing disorders due to faulty or ill-balanced food. I am still of the opinion that trachoma begins as adenomatous conjunctival irritation, due to improper feeding and impaired metabolism."

This is a fair expression of the opinion of those who believe that trachoma is a disease due to a lack of certain vitamins. My experience has caused me to believe that trachoma is a disease transmitted by infection and that it produces an infectious purulent secretion. I have seen many hundreds of cases develop as a result of contact with trachoma patients. These patients were not undernourished, but were healthy, well-fed children who were unfortunate enough to be placed in a school where trachoma was prevalent. Trachoma is now frequently referred to as "wrestler's disease" and we could hardly classify wrestlers as victims of malnutrition. I believe it is an infection conveyed from eye to eye by contact with the infectious secretion.

During a period of more than ten years, with my partner, the late Dr. George W. Hale, I had charge of the children in the Tennessee Industrial School. There were hundreds of cases in the school when I entered practice in 1899. I had just finished a service of nearly two years and a half at the Manhattan Eye and Ear Hospital, where I had seen a great deal of trachoma, but nothing to compare with the number and variety of the cases I found in the school. Our Sundays were spent in surgery at the school and a substantial part of our week days were taken up in local treatment at the office. A goodly portion of this work fell on me naturally. During this period I was startled by an outbreak of what

appeared to be an epidemic of purulent ophthalmia. About twenty of the little girls from ten to fifteen came down at one time with every appearance of purulent ophthalmia. There was intense swelling of the lids, great chemosis and profuse purulent discharge. The smears did not show the gonococcus, but the clinical picture was the same as that of gonorrheal ophthalmia. I isolated the children in a separate building and put nurses in charge to carry out the routine treatment of purulent ophthalmia. When we got to the bottom of the thing we found that the children had discovered that those children with infected eyes were carried into the city for treatment. This appeared to them quite an event in their drab lives and one enterprising youngster suggested inoculating their eyes with secretion from the eyes of the infected children. This was readily agreed to and was carried out with the dramatic results described above. The cases infected ran a stormy course, but terminated without corneal involvement and on subsidence of the acute symptoms settled down and ran the regular course of trachoma. To me this experience proved several things. No laboratory experiment could have demonstrated more clearly that trachoma is a transmissible disease carried by infectious secretion from one eye to another. It also showed that mass infection of trachomatous secretion reacts violently in the eye with all the symptoms of purulent ophthalmia. To my mind it also explains why we see few cases of acute trachoma. The amount of infection conveyed on towels and through the medium of wash basins is so small that the eye reacts to it mildly and it is only when the stage of folliculosis and granulation is reached that it attracts attention. In other words, the infectious agent is more or less feeble and where ordinary cleanliness is observed the chances of infection are slim. This would account for freedom from infection in families where there is a case of trachoma so that we may say that trachoma is a filth disease due to the indiscriminate common use of towels and wash basins.

DR. L. A. JULIANELLE, closing: I want to thank both Dr. Thygeson and Dr. Cullom for discussing this paper so graciously.

The thing that strikes me is that after all this discussion we have left trachoma in the confused state with which we started. I have not tried to define the cause of trachoma at all. I have merely attempted to discuss the various possibilities to be considered as inciting trachoma. It may very well be that the small rod-like structures Dr. Thygeson showed on the screen a moment ago actually cause this disease, but I do not feel that anyone has yet proved this. I hope that someone will bring sufficient evidence to prove conclusively whether the inclusion or elementary body does cause trachoma. On the other hand, it seems ridiculous to consider diet any longer when one realizes the large number of wrestlers who so readily become infected with trachoma. And during the past war, a number of prospective recruits were able to avoid service by deliberately infecting themselves from recruits rejected because of trachoma.

I have no more to add to what I have already said, because I am trying to take a liberal attitude. I am not trying to define the causative agent of trachoma; I merely want to report on a new method of study which may help in arriving at a conclusion more rapidly than otherwise.

THE TRACHOMA SITUATION IN SOUTHERN ILLINOIS

H. S. Gradle, M.D., A. F. Lenzen, M.D.
and A. M. Hayden

CHICAGO

During the early part of the preceding century, settlers from the Eastern Seaboard migrated westward to what was then called the Western Province of Virginia. They settled around the junction of the Ohio and the Mississippi Rivers and spread out into the hills of Kentucky and on to the plains of Missouri and Illinois. The descendants of these early settlers still form the main portion of the population of this territory, as the foreign emigration passed by this part of the country. When trachoma first made its appearance in this area is unknown, but ophthalmologists have known of the existence of the disease there for at least forty-five years. The late Dr. Stucky started the campaign in the Kentucky hills and his early efforts are being carried on in the endeavor to control the scourge there. In Missouri, the United States Public Health Service is fighting the disease and apparently gaining the upper hand.

In Illinois, trachoma is fairly well confined to the southern fourteen counties of the state. How many cases there are cannot even be conjectured, for no comprehensive survey has ever been made. In four of the counties, the Illinois Society for the Prevention of Blindness made two surveys, spaced ten years apart, and found that the number of cases had not decreased in the single decade. No effort, either private or governmental, had ever been made to alleviate the conditions there or to prevent the spread of the disease, and it was rather evident that the wave of trachoma was advancing slowly toward the north. There were no eye clinics in the southern end of the state and a few of the worst cases were sent to the Eye and Ear Infirmary in Chicago, a most unsatisfactory method of procedure, on account of the distance of that institution from the trachoma regions and the consequent homesickness of the patients.

These facts were laid before Governor Horner shortly after his inauguration and he took immediate steps to finance a campaign for the control of trachoma in the southern counties. The Illinois Society for the Prevention of Blindness contributed most generously, both in money and personnel. The State Board of Health and the Department of Public Welfare, particularly through the Illinois Eye and Ear Infirmary, contributed their quota of work, and on June 18, 1934, the Governor Horner Trachoma Clinics were opened. At this time, the Illinois Emergency Relief Commission was functioning and, due to the splendid cooperation of this body, some of the nurses and nearly all of the non-technical personnel connected with clinics were furnished as a relief project.

Reference to the accompanying map will furnish a geographical concept of the territory covered, and of the counties that were served by the clinics. Outpatient clinics were started in Harrisburg, Shawneetown, Marion and Jonesboro. Space was provided by the local authorities in the public libraries or the

courthouse, so that each clinic had a waiting room, a treatment room and an operating room. Uniform equipment was placed in each clinic so that the personnel could function smoothly in any one of the four locations. Two nurses were assigned to the two eastern clinics and two nurses to the two western clinics, all under the supervision of Dr. Lenzen. Each clinic was operated for one full day each week, with the physician and three nurses on duty, and for one-half day with the same personnel, and for one-half day with only one nurse. The remaining time of the nurses was spent in treatments and home visits. In addition to the medical personnel, each clinic had the service of stenographers, social service workers, janitors, and scrubwomen. Operations were performed in the clinics on given days by appointment.

But little publicity was required, for news of the establishment of the clinics spread rapidly by word of mouth and the clinics were soon operating at capacity. Each new case was questioned very carefully, not only as to medical history, but also in regard to the social status. Great care was exercised not to accept patients who were under the care of private physicians or patients whose financial condition was such that they could engage the services of a private physician. Of course, only cases with trachoma were accepted for treatment. All others who reported to the clinics were examined carefully and either released as non-trachomatous, or else listed as trachoma suspects and re-examined at a future date. Surveys were made of several of the schools as possible nests of infection and all children with trachoma were referred to the clinics for treatment. Careful records, both medical and social, were kept in every case and this paper is based upon an analysis of those records. The period of this report covers from June 18, 1934, to June 18, 1935.

Total number of Positive cases of Trachoma was 933. Of these, 549 were males and 384 were females.

The ages of the patients were as follows:

From 1 to 9 years inclusive	98	
From 10 to 19 years inclusive	151	415 or
From 20 to 29 years inclusive	90	44.5%
From 30 to 39 years inclusive	76	
From 40 to 49 years inclusive	152	
From 50 to 59 years inclusive	164	
From 60 to 69 years inclusive	115	
From 70 to 79 years inclusive	73	
From 80 to 89 years inclusive	14	
TOTAL	933	

The length of time that the condition had existed in each case previous to admission is of import, both from the preventive standpoint as well as the therapeutic. For such information, the word of the patient had to be relied upon and as nearly as could be estimated, the following condition pertained:

Trachoma present for 1 month or less	92 eyes	304 or 32.5%
Trachoma present for 2 to 6 months inclusive	82 eyes	
Trachoma present for 7 to 12 months inclusive	130 eyes	
Trachoma present for more than 12 months	1298 eyes	
No record of length of time condition existed	264 eyes	
TOTAL	1866 eyes	

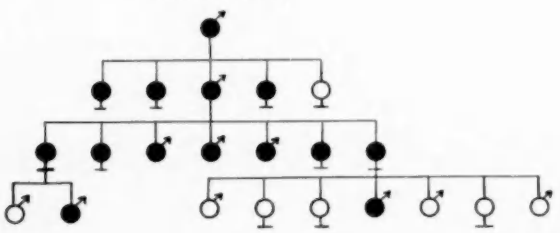
Four hundred ninety-seven of these patients had had previous treatment elsewhere. It was not only of interest, but also of great importance to determine the number of members of each household from which a patient came who were afflicted with trachoma.

MEMBERS OF HOUSEHOLD AFFECTED

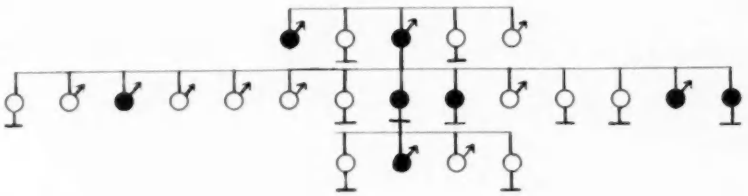
1 member affected	631 cases
2 members affected	169 cases
3 members affected	81 cases
4 members affected	31 cases
5 members affected	9 cases
6 members affected	6 cases
8 members affected	6 cases

In this connection, it may be of interest to reproduce two family trees showing the number of cases of trachoma through the different generations. The black circles indicate the members infected with trachoma.

FAMILY G.



FAMILY R.



In order to simplify the keeping of records, we classified the conditions of the trachomatous conjunctivæ as

STAGE 1 — Acute, succulent, purulent, follicular, granular.
STAGE 2 — Hypertrophic, hyperemic.
STAGE 3 — Beginning scar formation with chronic inflammation.
STAGE 4 — Quiescent scarred condition.

It was, of course, impossible to classify each case accurately as one stage is apt to overlap into another, but holding to the classification as accurately as possible, the following was found on admission:

Stage 1	834 eyes	928 or 49.7%
Stage 2	94 eyes	
Stage 3	55 eyes	
Stage 4	795 eyes	
Unilateral Trachoma	24 eyes	
No record	64 eyes	
TOTAL	1866 eyes	

The fact that the disease had existed for such a long time in so many of the cases was responsible for the large number of complications that were found upon admission.

Trichiasis existed in	483 eyes
Entropion was present in	225 eyes
Pannus had formed in	905 eyes
Active corneal ulcers were present in	35 eyes
Corneal scars were present in	441 eyes
TOTAL COMPLICATIONS	2089 eyes

Inasmuch as the preventive phase of the work was to be emphasized as well as the therapeutic, it was of import to have as accurate a record as possible of the visual acuity upon admission to the clinics as well as upon completion of the treatment. All visual acuities were measured at a twenty foot distance with a standard test chart and minimum illumination of seven foot candles.

Vision of 20/20	565 eyes	
Vision of 20/30 to 20/40 inclusive	293 eyes	
Vision of 20/50 to 20/60 inclusive	118 eyes	
Vision of 20/70 to 20/100 inclusive	291 eyes	
Vision of 20/200	143 eyes	
Vision of less than 20/200	375 eyes	
Impossible to obtain vision	45 eyes	22%
Amaurosis	36 eyes	
TOTAL	1866 eyes	

It should be noted that the number of eyes with vision of less than 20/200 and amaurotic eyes total 411, or *exactly 22% of the total.*

Nine hundred thirty-three patients were diagnosed as trachomatous during the year covered by these records. Of that number, 12 were referred to private physicians because of their economic status and were given no treatment in the clinics. Three were too ill to come to the clinics after the first visit and were not seen again. In 1 case, both eyes had been enucleated and the conjunctivæ were such that no treatment was advised. That left 917 patients that were given treatment of which there were ten main types. The greatest reliance was placed upon chalmoogra oil massage, but in some it was combined with the use of other medication. A few cases were treated with quinine bisulphate alone and in some it was combined with zinc ionization. Others were treated with glass rod massage. Every patient was given a 1-5000 aqueous solution of oxycyanide of mercury to use as an eyewash two or three times daily at home in addition to the treatments given in the clinic. Considerable stress was laid upon that as a preventive of secondary infection. To summarize, the 12,092 treatments that were given in the clinics were divided as follows:

	Patients
Chalmoogra massage alone	474
Chalmoogra massage plus Quinine Bisulphate	33
Chalmoogra massage plus Quinine Bisulphate plus Zinc ionization	3

Chalmoogra massage plus Zinc ionization	14
Quinine Bisulphate alone	11
Oxycyanide of Mercury alone	346
Glass rod massage	6
Finger massage	1
Copper Sulphate	1
Mixed Treatment	28
TOTAL	917

The maximum number of treatments given any one patient was 115. No reference will be made here to the comparative value of the various forms of treatment as that will form the basis of another paper of a non-statistical nature.

Operations were performed in the various clinics as requisite, using both local and general anesthesia, as follows:

	No. Eyes
Expression	112
Modified Ewing	69
Hotz	39
Canthoplasty	46
Cautery puncture	30
Spencer-Watson	5
Gaillard Suture	11
Grattage	44
Lip graft	3
Diathermy Peritomy	10
Electrolysis of lashes	11
Pterygium	14
Chalazion	11
Tearsac Extirpation	1
Excision of Scar Tissue	1
Sling Suture of Outer Canthus	1
Excision of Multiple Calculi	10
Tearsac Puncture	1
Incomplete Expression	9
Entropion	1
Excision of Sebaceous Cyst	3
Cilia Removed	1
Plastic Operation on Canthus	1
Plastic Operation	1
Forcible Dilatation of Tear Passages	1
TOTAL NUMBER OPERATIONS	436

The final vision was recorded in 478 cases. Accepting the viewpoint that vision of 20/50 or better is required for satisfactory reading ability and that 20/200 or better is required for

ordinary labor, the improvements in vision that followed treatment may be classified as follows:

Vision became worse during treatment	48 eyes or	5.02%
Vision improved from less than 20/50 to 20/50 or better	96 eyes or	10.04%
Vision improved from 20/200 or less to between 20/60 and 20/200	67 eyes or	7.00%

The economic status of the patients is a matter of more than passing interest and according to the best information possible the new patients at the clinic for the past year fell into the following classes:

486 were on relief, of which number
- 465 were getting relief from the IERC
- 10 were in CCC camps or supported by someone in camps
- 6 were on the Blind Pension Roll
- 5 were receiving War Pensions

284 were self supporting
45 were subsisting on farms, but without any ready cash
12 refused information as to their means
15 had no income and were living with relatives
91 were in the unclassified list

933 TOTAL

In the self-supporting group of 284, the average weekly income for the family was $10.05, the maximum being $26.00. The size of that average family was 4.63 individuals, which yielded an average individual weekly income of $2.17. In this connection, it might be of interest to note that the Blind Pension Roll of the fourteen counties that are served by these clinics contains 615 names. Inasmuch as the statistics of the Blind Pension are of no value from the scientific standpoint, it is impossible to say how many of these cases are blind due to trachoma.

The territory served by these clinics is distinctly rural, with scattered small towns. It was found that of the 933 patients seen in the first year of the clinics, 183 lived in the communities where the clinics were held. The remainder lived at varying distances away, the average being 16.1 miles and the maximum 56 miles. Thus, the transportation problem for these patients is a serious one. Many are hauled in by friends. Some come in buses that the IERC operates. The majority hitch-hike or walk. For example, one family with four trachomatous members, lived 22 miles away from the nearest clinic. A of that family came for 32 treatments, B for 3 operations, C for 45 treatments, and

D for 54, and every mile of that distance was covered on foot. Innumerable such examples are available, but do not lend themselves to any statistical analysis.

Summary

On June 18, 1934, four outpatient clinics for the care of indigent sufferers from trachoma were opened across the southern end of Illinois. These were made possible through the understanding and generosity of Governor Henry Horner.

During the first year, 933 cases of trachoma were diagnosed. Four hundred fifteen of these cases were under 40 years of age, indicating the necessity for a long-continued campaign.

Of the 1,866 eyes seen, the condition had existed for one year or less in 304 eyes, the remainder being more than a year old.

In 302 cases, more than one member of the household had trachoma.

The number of eyes in the various stages of the disease and the complications that existed upon admission are enumerated.

Twenty-two per cent of the total number of eyes had less than 20/200 vision.

The methods of treatment and the total number of treatments are analyzed, but without discussing end results.

The visual end results of 478 cases with completed records were analyzed with the finding that 5.02% of the eyes lost some vision despite treatments; 10.04% of the eyes improved to 20/50 or better; and 7.00% of the eyes improved to 20/200 or better.

Only 329 of the patients were self-supporting and the remainder were dependent upon some form of charity. The average income of the self-supporting patients was $10.05 per week for the entire family that averaged 4.63 individuals.

DISCUSSION

Dr. Richard C. Gamble, Chicago: About twenty-five years ago, Dr. Stucky wrote a very dramatic chapter in American ophthalmology. He was impressed by the number of patients with trachoma who came to him from the mountain regions of Kentucky. His interest in the problem finally became so great that he went into the hills himself and found that trachoma was literally a scourge among the native white population. There were few doctors, no hospitals and no roads by which the afflicted persons could go to seek medical attention. It was obviously necessary to bring medical attention to the patients. He did this in an inspiring manner and the work he started is being continued by the Public Health Service, not only

in Kentucky, but also in Missouri and on Indian reservations. A similar situation has long existed in the southern part of Illinois, and it is gratifying to know that Dr. Gradle, Dr. Lenzen, Miss Hayden and their organization are handling the situation so adequately.

It is very interesting to compare this report with the reports of similar campaigns in Kentucky twenty-five years ago. Stucky in 1913 examined 4,000 persons in five counties in Kentucky and found that 12.5% of them had trachoma. It was estimated by Gordon Berry that there were 33,000 persons infected with trachoma in Kentucky in 1915. Among the 323,000 Indians in the United States at that time, 65,000 or 20% were estimated to have trachoma. The comparative figures on the prevalence of complications and sequelæ and the frequency of visual loss are of even greater interest. Stucky found that 40% of the trachoma patients had corneal complications, 10% had entropion and 25% had impaired vision. Gordon Berry found that in 1,000 consecutive cases of trachoma treated by the U. S. Public Health Service in 1915, there was corneal opacity in 24.5%, corneal ulcer in 6.4%, pannus in 28%, entropion in 13.9%, and trichiasis in 10.8%. Vision was impaired in 50.2% and 5% were blind. Among the 933 patients reported in this present paper, there was corneal opacity in 24%, corneal ulcer in 1.8%, pannus in 48%, entropion in 12% and trichiasis in 25%. The vision was less than 20/50 in 45% of these patients, and in 22% it was less than 20/200. From consideration of these figures one is forced to conclude that trachoma in southern Illinois at the present time is a more serious disease than it was in Kentucky twenty-five years ago. This is a serious situation and is a challenge to those working in this field to carry on.

There are several other minor points which perhaps deserve comment. There were fewer patients between the ages of 20 and 40 years than there were in the other groups, excepting, of course, young children and the very old. This, perhaps, is due to the fact that persons in the third and fourth decade of life frequently leave the farm and seek employment in the factories in the larger cities. One is also impressed by the large number of patients who were listed as having Stage 1 and Stage 4 of the disease and the small number having Stages 2 and 3. In regard to the methods used in treatment, it is interesting to note the fact that in no case was the tarsus removed. This is quite different from the questionable practice in many trachoma clinics where tarsectomy has been almost a routine procedure. It is quite surprising to find that only one patient was treated with copper sulphate alone. Possibly some of the 28 listed as receiving mixed treatment were treated in part with copper sulphate, but even so the number was a very small percentage of the total number of patients. The long and frequent trips many of the patients have to make to the clinic must be an arduous undertaking. If treatment with the copper sulphate stick in the clinic and copper sulphate in glycerine at home, instead of chalmoogra oil, would reduce the need for frequent visits to the clinic it might be preferable, at least in the case of patients who live far from the clinics.

Dr. Gradle, Dr. Lenzen and Miss Hayden are to be congratulated for bringing this interesting paper to our meeting and for doing a splendid piece of work for the citizens of the State of Illinois.

Section on Otolaryngology

The American Academy
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Research Report on
EXPERIMENTAL AND CLINICAL SINUSITIS*

Ralph A. Fenton, M.D., and
Olof Larsell, Ph.D.
PORTLAND, ORE.

SYNOPSIS OF EXPERIMENTS

During 1934 and 1935 we continued our previous study (1930-1934) of the effects of surface applications to the normal and to the inflamed frontal sinus membranes of the cat, in which acute or chronic inflammatory changes closely resembling human processes had been produced by inoculation with human strains of hemolytic streptococci. Technic and subsequent preparation of tissues were the same as that heretofore employed.

Study of the absorption of particulate matter into the sinus membranes was carried on by chipping away the bony sinus roof and placing 5% prussian blue in 4% gelatin, and in other animals full-strength india ink, outside the intact normal membrane. It was found that numerous histiocytes were attracted to the region exposed to the ink and the prussian blue; these remained crowded together for 24 hours, were spread out for some distance in 48 hours, and very widely scattered and thinned out over the whole sinus roof in 72 hours. By this method, no distinct accumulation of ink-laden histiocytes within definite lymph channels could be demonstrated; but the immediate defense reaction of tissue phagocytes (histiocytes) was readily visible, even under low power.

Reaction of cat membranes to histamine was tested as follows: by injection of $\frac{1}{1000}$ mg. of histamine in solution outside an intact normal sinus membrane and in 4% gelatin into a normal sinus cavity; and by perfusing a 6 weeks infected sinus and a normal sinus three times 24 hours apart with a solution of $\frac{1}{1000}$ mg. histamine to 1 cc. sterile distilled water. This agent produced some thickening of the tunica propria, used outside; and great

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distension of the goblet cells, used inside the normal sinus. In the chronic infected membrane very severe acute exacerbation was produced by repeated doses in the sinus, but merely some edema, without mucosal damage, in normal membrane.

Azochloramid, a chlorine-releasing antiseptic in buffered 1:2000, 1:5000, and 1:10,000 solutions, was perfused through normal, and acute and chronic infected sinuses; no reparative effects were noted after 72 hours, and the stronger solutions seemed to be definitely destructive to the mucous membrane.

Glycin (glycocoll) 2.5% isotonic solution, because of its reported influence upon the autonomic nervous mechanism, perfused through normal and chronic infected sinuses, produced no reparative effect except an increase in number of goblet cells.

Acetylcholine (2.5 mg. to 5 cc. sterile distilled water), which has been much advocated for ozœna because of its stimulating effects upon vasodilatation, caused congestion of the normal and very marked edema of the chronic infected membrane, without evidence of recent leukocytic migration or other repair.

Amniotic fluid, used to promote defense reactions in the peritoneum, perfused half- and full-strength through acutely infected sinuses, at 48 hours showed very marked round cell infiltration and extravasation of blood, with great epithelial damage. After 72 hours, in chronic infection, changes were observed, with much less infiltration than that usually found in chronic infection; apparently decided benefit.

Chlorophyll in an isotonic solution was found irritating in acute infections, but stirred up moderate exacerbation of sinus inflammation.

Sodium alum 1% and tannic acid ½% solutions, perfused through sinuses which were inoculated ten minutes later with hemolytic streptococci, seemed to inhibit decidedly the severity of the acute infection.

Exposure of chronic infected sinus membranes to the bare quartz light for ½ to 1 minute did not seem to produce any reparative changes after 72 hours.

Production of absolute constipation during 120 hours in order to promote absorption of intestinal toxins into chronic infected sinus membranes did not give rise to an acute exacerbation of the sinusitis.

Ten new compounds thought to have effects similar to ephedrin were perfused through acutely infected sinuses in 1%

solution. All showed rather marked leukocytic infiltration; *d-l*-methylephedrine methiodide showed many eosinophils, especially in the blood vessels, and numerous plasma cells. Isoamyldimethyl amine methiodide showed marked infiltration with macrophages, plasma cells, leukocytes, and some histiocytes. Similar findings were noted with benzyl dimethyl amine methiodide and hordenine methiodide. The most marked subepithelial reparative effect was found with tetrandrine methiodide.

Our experiments of 1934 on unilateral cervical sympathectomy in chronic sinus inflammation were repeated, with the same results then reported: severe exacerbation and engorgement on the sympathectomized side.

Study of tissues from infected antrum mucosa and cyst walls, from a man of 36 who had received heavy doses of deep roentgen rays (because of suspected malignancy), showed enormous thickening and fibrosis of the tunica propria, many plasma cells, and almost normal epithelium.

Use of isotonic azochloramid solution 1:5000 and 1:10,000 proved somewhat irritating to the nasal mucosa of eleven patients when used for antrum irrigation, provoking some swelling and sneezing. While it reduced the odor in foul cases, it did not seem to reduce the amount of pus especially, and was discontinued because of its irritating qualities.

Isotonic chlorophyll solution, injected into human antra because of its stated properties of furnishing bacterial nourishment and thus freeing the infected membrane from attack, apparently increased rather than diminished the amount of exudate and odor in eight chronic cases; in two, slight improvement was noted during its use. No complaint of irritation was made.

Much clinical improvement and cessation of discharge was noted in two chronic antra into which amniotic fluid full-strength was injected several times following ordinary lavage. No discomfort was experienced, but the curative effects of the lavage may have been responsible.

Quantities of the new ephedrin-type compounds were insufficient to try them in human sinuses, but it was found that in 1% solution all produced some shrinkage and blanching when applied to the inferior turbinate, especially: isoamyl dimethyl amine methiodide, benzyl dimethyl amine methiodide, tetrandrine methiodide, beta-phenyl ethylephedrine methiodide.

We are very grateful to the Research Committee and the Council of the American Academy for their continued and generous support of this research.

Sincere thanks are tendered to Dr. Franz Schmelkes of Wallace & Tiernan, Inc., for the supply of azochloramid; to Drs. R. F. Ridpath and Benj. Gruskin, Temple University, for the supply of and information regarding chlorophyll solution; to Dr. K. K. Chen of the Lilly Research Institute for the supply of new ephedrin-resembling compounds; and to Dr. Charles Armstrong, U. S. Public Health Service, for information regarding the inhibitory effect of astringent drugs upon subsequent mucosal infections by viruses.

RECAPITULATION

Five years of investigation into the histopathology of experimentally produced sinusitis, with clinical applications of certain data thus secured, have convinced us that, owing to the defensive factors inherent in sinus epithelium and the connective tissue elements of its tunica propria, almost every sort of surface application to such membranes becomes an irritant unless its strength be isotonic. Solutions stronger than normal are almost certain irritants, as are those which in any way interfere with ciliary action, no matter how well they may work as test tube antiseptics. Only a few chemical substances are favorable to cell growth and stimulate an influx of reparative histiocytes and leukocytes. An index of various substances and agents used during all these researches, with their results, is annexed to this report; also a note detailing the technic of diathermy for sinusitis as reported last year.

This year we have demonstrated that histiocytes take up particulate matter from the outer (bony) side of the sinus membrane as well as from the lumen of the cavity, and that such matter is carried widely through the subepithelial tissue within 72 hours of its introduction, pointing to the likelihood of similar dissemination of bacteria in histiocytes.

Histamine in the normal sinus merely increases the flow of mucus; but apparently its presence in repeated doses causes severe acute exacerbation of chronic suppurations. We were not, however, able to produce this exacerbation by causing complete arrest of fecal elimination for five days; evidently in the cat not sufficient increase in histamine occurs under such cir-

cumstances through absorption of intestinal toxins to cause any exacerbation of existing sinus lesions.

A new chlorine-releasing antiseptic, azochloramid, was found to share the irritant properties of similar substances previously reported upon, without stimulation of repair.

No reparative effects were noted from surface application of glycin or acetylcholine, although the latter brought about marked edema of both acute and chronic infected membranes. Chlorophyll solution caused severe acute exacerbation of chronic inflammation without reparative effects.

Amniotic fluid, half- and full-strength, caused marked congestion, leukocytic infiltration and early fibrous reparative changes, with diminution of the superficial destructive changes during 72 hours.

Astringents (sodium alum and tannic acid) used in weak solution inhibited very greatly the severity of infection when such sinuses were subsequently inoculated.

Ten new ephedrin-resembling compounds were all found to have slightly stimulating effects toward repair, notably isoamyl dimethylamine methiodide, benzyl dimethylamine methiodide, hordenine methiodide, and tetrandine methiodide. All of these produced slight blanching and shrinking of human mucous membranes, especially the latter three.

Proof of the excessive fibrosis caused by roentgen ray therapy was afforded by tissues from a man operated upon for severe maxillary suppuration with cyst formation, who had received heavy dosage for suspected malignancy.

Azochloramid 1:5000 isotonic solution was found too irritating for antrum lavage. Chlorophyll in isotonic solution proved helpful in only two out of ten chronic antrum cases. Much clinical improvement was noted in two cases in which amniotic fluid was used after lavage.

We do not feel that further histopathologic study of cat sinus membranes treated by the surface application of solutions will be profitable at present. Perhaps biochemists, physiologists and bacteriologists with more extensive equipment and opportunities may find it possible further to develop some of the lines of investigation which have opened before us during this and preceding years.

The anatomical problems of lymphatic drainage of the sinuses, and of their autonomic innervation, remain as yet unsolved.

These are difficult and delicate problems, but are nevertheless of much importance in the interpretation of sinus physiology and pathology.

DETAILS OF EXPERIMENTAL STUDIES

The experimental work was carried out with the technical assistance of Mr. Wallace Shearer, in the Department of Anatomy. Previous inoculation technic and staining were followed, except that vital staining with trypan blue was not done on cats 35-1 to 35-12, inclusive.

Spread of Particulate Matter Outside Sinus Membrane — A series of normal cats had the bone chipped away over the intact frontal sinus membrane, upon which (cat 35-1) was deposited 5% prussian blue in 4% gelatin, membrane secured immediately; full-strength india ink: cat 35-2, membrane secured after one hour; cat 35-3, after 24 hours; cat 35-4, after 48 hours; cat 35-5, after 72 hours. These membranes were lightly stained with hematoxylin eosin, spread out and mounted full thickness on large slides. Very little absorption was noted with the prussian blue. After one hour the black mass was found surrounded by histiocytes already laden with ink. The spread of these ink-laden histiocytes did not seem to follow definite channels, but they became more and more widely dispersed up to 72 hours, while the central mass grew progressively smaller. Thus particulate matter, and by analogy histiocytes bearing infectious matter which they may ingest from some focus, may be spread within 72 hours through a large portion of the tunica propria without any lesion of the epithelium.

Use of Histamine — Cat 35-6. 1/1000 mg. histamine dissolved in water was injected on the outer surface of the intact right sinus membrane exposed by chipping away the bony wall, and the skin wound sutured. This dose was repeated after 24 hours. 1/1000 mg. histamine in 4% gelatin was injected into the left sinus cavity, repeated after 24 hours. Membranes were secured 72 hours after the first injections. No systemic reaction took place. With the outside injection, slight thickening of the tunica propria and some cellular infiltration occurred. The epithelium is thickened, goblet cells are numerous but not distended, and cilia are partially destroyed. Injection into the sinus shows a normal membrane except for distended goblet cells.

Evidently this quantity of histamine is definitely irritating to the tunica propria, but not to the epithelium.

Cat 35-8 was inoculated with hemolytic streptococci in the right frontal, and developed a purulent rhinitis which lasted five days after inoculation, later sneezing occasionally during six weeks, after which interval three injections of $\frac{1}{1000}$ mg. histamine in 1 cc. distilled water were made into the right (infected) and left (normal) sinuses, 24 hours apart. Membranes were secured 72 hours after the final injection. The cat was noticeably sick and irritable after the second histamine injection. The right sinus, quiescent at the first injection, was filled with purulent exudate 24 hours later. Following the second injection, the right side became so violently inflamed that pus exuded from the needle wound and appeared on the scalp. The third injection further aggravated both the local and general conditions of this animal. Grossly, the right sinus was full of a necrotic mass, while the left sinus membrane was merely somewhat congested and adherent to the bony wall at the site of injection. Microscopically, the right membrane showed greatly thickened tunica propria with edema and post-infectious fibrosis. Near the epithelial surface marked acute exacerbation of inflammation had occurred, with leukocytic infiltration, hemorrhage and destruction of epithelium. The left membrane showed little change except for accumulation of fluid in the tunica propria, epithelium remaining normal.

Use of Azochloramid — This chlorine-releasing antiseptic was used in buffered isotonic solutions, perfused through the injection needle until the solution ran out of the nostril, after which membranes were secured in 72 hours. Cat 35-9, chronic infection (6 weeks) right sinus, normal left sinus, showed no general effects from use of 1:10,000 solution. Grossly, the usual chronic thickening of the right membrane was observed. Microscopically, there was no evidence of an acute reparative effect in either membrane, the left being quite normal. Cat 35-11, right chronic, left normal, perfused with 1:5000 solution, seemed somewhat depressed after the injection. Grossly, the chronic membrane was thick but not adherent, with some purulent exudate in the sinus cavity. Microscopically, in addition to the usual chronic thickening and edema, there was acute injury to the epithelium, but without reparative leukocytic infiltration. The uninfected membrane showed very severe epithelial de-

struction, thickening and injection, with recent fibrosis extending through the epithelium. Cat 35-17, acute infection left frontal, perfused with 1:800 solution: Grossly, this membrane was thickened, congested but not adherent, with sinus cavity filled with purulent exudate. The acute infection had caused edema and round cell accumulation, with some leukocytes and plasma cells and early fibrosis; but the epithelium was much injured and congested. Apparently this agent is an epithelial irritant and interferes with the normal process of repair, particularly in higher concentrations.

Use of Glycin — Cat 35-10, right chronic, left normal, was perfused with an isotonic (2.5%) solution of glycin (glycocoll). No systemic reaction occurred. After 72 hours, the chronic membrane showed the usual gross thickening without exudate, and the left was normal. Microscopically, no evidence of any reparative effect from the injection could be made out, except for a large number of goblet cells.

Use of Acetylcholine — Cat 35-12, right chronic, left normal, perfused with 2.5 mg. acetylcholine* in distilled water, membranes secured after 72 hours. This animal seemed much depressed after injection, especially the last 24 hours. The chronic membrane was thick, congested, adherent and full of necrotic exudate; the normal membrane was engorged with blood at the site of injection. Microscopically, the chronic membrane was greatly congested, edematous, but with little or no leukocytic infiltration; the epithelium was damaged, cilia gone in patches, and numerous goblet cells. The uninfected membrane showed some thickening and distinct congestion of the tunica propria, with epithelium normal save for numerous large goblet cells and some ciliary damage.

Use of Amniotic Fluid — Cat 35-13, both frontals acutely infected, perfused with amniotic fluid† full strength right, diluted half with normal saline solution left. This animal had a severe reaction from its streptococcus inoculation 48 hours before, and was much depressed when anesthetized for the amniotic fluid perfusions, and died 20 hours after the latter. Both membranes were severely congested and the sinus cavities were full of necrotic exudate. Microscopically, there was very great infiltration of both membranes by lymphocytes, with much extrav-

* Commercial acetylcholine used was "Acecholine."
† Commercial amniotic fluid used was "Amfetin."

asation of blood and destruction of epithelium. Cat 35-15, both frontals chronic, right perfused with amniotic fluid full strength, left half strength, membranes secured at 72 hours showed general reaction. Grossly, both membranes were somewhat thickened and adherent, with a little purulent exudate in the right and less in the left cavity. Microscopically, aside from the usual chronic thickening, and plasma cells, the tunica propria shows some active fibrosis and little damage is apparent in the epithelium; apparently the injections were distinctly beneficial in these chronic membranes.

Use of Chlorophyll — Cat 35-16, chlorophyll buffered isotonic solution (Gruskin) 2 cc. injected into left acutely infected frontal, membrane secured at 72 hours, somewhat thickened with moderate amount of purulent exudate. Microscopically, very great thickening, hyperemia and polymorphonuclear infiltration, with ciliary destruction and much epithelial invasion by leukocytes; apparently a very severe exacerbation of the acute inflammation with no evidence of repair, possibly due to the solvent used for the chlorophyll. Cat 35-19, chronic infection, right sinus failed to receive injection, left sinus 2 cc. chlorophyll injected; grossly, much thickened after 72 hours, still containing the green solution mixed with more purulent exudate than that found on the right side. Microscopically, marked thickening and edema with leukocytic infiltration and numerous plasma cells were found; apparently an acute exacerbation due to the solution, but without much epithelial loss or reparative stimulation.

Prophylactic Effects of Sodium Alum and Tannic Acid — Cat 35-14, not infected, was perfused, right frontal with ½% tannic acid solution, left frontal 1% sodium alum solution; ten minutes thereafter each sinus was inoculated with hemolytic streptococcus. There was a slight mucopurulent discharge from both nostrils but the cat was not sick at any time. After 72 hours the membranes were secured. A small amount of purulent exudate was found in each cavity; the membranes were not swollen or congested. Microscopically, with both prophylactic agents, there was slight thickening and leukocytic infiltration of the tunica propria, but very much less than in membranes untreated prior to inoculation. The cilia were slightly damaged but epithelium was otherwise normal. Both sinus cavities were filled with pus.

Effects of Quartz Light — Cat 35-18, bilateral chronic infection, had the sinus roof chipped away and the right sinus membrane was irradiated with a standard quartz tube point within the cavity for one minute; the left membrane for 30 seconds. The scalp was sutured and membranes secured after 72 hours. Both sides were thickened and the membranes were very adherent; some purulent exudate was found on the right side. Apparently the chronic infection had been more severe in the right sinus. Microscopically, there was marked polymorphonuclear infiltration on the right side, probably a secondary infection. None of the acute changes characteristic of a reparative process were found.

Effects of Chronic Constipation — Cat 35-20, bilateral chronic infection, was fed 0.1 gm. sodium acid tartrate daily for one week, causing loose grayish stools after the fifth day. The eighth day 5 mg. powdered opium was given, repeated until the eleventh day, when the dose was increased to 10 mg. for four more days. Bowel movements ceased after the third day of opium, and after 120 hours of constipation during which full meals had been consumed, the membranes were secured. The entire large intestine was filled with fecal material. Grossly, the sinus membranes were adherent, containing a little purulent exudate, but presented no signs of acute irritation. Microscopically, the usual chronic changes were observed, with plasma cells and some fibrosis, normal epithelium, and no evidence of acute exacerbation from any source.

Effects of Drugs Resembling Ephedrin on Acute Infection — Cat 35-21, acute bilateral, right sinus perfused with 1% methylephedrine methiodide; left with 1% *d-1*-methylephedrine methiodide. Animal was depressed following injections. All membranes in this series were secured after 72 hours. The left side was more thickened, both much congested. Microscopically, both sides showed much epithelial destruction and acute infiltration, but the left showed numerous eosinophils, especially in the blood vessels. Cat 35-22, acute bilateral, small sinuses deep in cancellous bone, which became infected. Right side could not be perfused with 1% beta-phenyl-ethyl dimethyl amine methiodide, which had apparently no effect in arresting the infection about the sinus in the cancellous tissue. Left sinus was perfused with 1% *d-1*-pseudo-methylephedrine methiodide. Its membrane was thick, adherent and contained pus. Microscopically, there was

marked thickening, fibrosis and edema, with leukocytic infiltration and epithelial destruction. Cat 35-23, acute bilateral, perfused right sinus 1% isoamyl dimethyl amine methiodide; left with 1% *p*-phenyl-beta-hydroxy ethyl dimethyl amine methiodide. The infection was severe, animal much depressed, membranes greatly congested and full of pus, more on the right side. Microscopically, right side, severe acute infection, marked epithelial destruction, tunica propria filled with macrophages, plasma cells and leukocytes. Left sinus, infiltration and reparative effects less marked. Cat 35-24, acute bilateral, right sinus perfused with 1% benzyl dimethyl amine methiodide, left with 1% hordenine methiodide; animal unaffected by experiment, slight nasal discharge when perfused, membranes not adherent, little exudate. Microscopically, right sinus, marked lymphocytes, plasma cells and some histiocytes; epithelium almost destroyed. Left sinus, marked infiltration by plasma cells, lymphocytes and some histiocytes; blood vessels engorged and epithelium full of leukocytes and nearly destroyed. Cat 35-25, acute bilateral, right sinus perfused with 1% tetrandrine methiodide, left with 1% beta-phenyl-ethyl ephedrine methiodide. Animal was somewhat depressed during experiment. Right sinus membrane was decidedly congested, containing much pus. Left membrane not congested, little exudate. Microscopically, right side, membrane thickened, barrier of leukocytes, especially, just below and in epithelium, which was somewhat injected; some plasma cells and histiocytes in deeper layers of tunica propria. Left membrane showed less marked reparative effects with little epithelial damage.

Effects of Unilateral Cervical Sympathectomy — Cat 35-7, acute bilateral, developing nasal discharge second day, was given 2 cc. per kg. body weight of an 11% solution of sodium barbital 72 hours after inoculation. Under complementary ether anesthesia the right cervical sympathetic was severed; membranes were secured 48 hours thereafter. Both sinuses were filled with purulent material, but on the right side the membrane was very much thicker and pus was oozing out through the needle wound. This confirms, on acutely infected membranes, our findings of last year respecting exacerbation caused by sympathectomy on chronic infection of the sinus wall.

Effects of Deep Roentgen Ray Therapy on Human Maxillary Mucosa — Tissues secured by Caldwell-Luc operation from the maxillary sinus of a man of 36 who had elsewhere received

several courses during five months of deep roentgen ray therapy for suspected malignancy diagnosed from roentgenographs, showed extreme toughness and adhesion of the mucosa and cyst walls to the dry and ischemic maxillary bone. Microscopically, the epithelium was about normal, but the tunica propria was excessively thickened and fibrotic, with but few infiltrating cells save some subepithelial eosinophils. A cyst wall from this case was highly edematous and fibrotic, with numerous plasma cells and normal epithelium.

APPENDIX I

Technic Employed for Treatment of Sinuses by Diathermy — Because of numerous requests, the following details are supplied by Dr. Arthur C. Jones, who administered these treatments in our 1934 series of cases: "The current is applied to the frontal and maxillary regions through a set of electrodes shaped to fit smoothly over the sinuses. (Medium weight electrode foil is used.) The electrode for the forehead is ovoid, about 1x2½ inches, with corners well rounded. A small strip connects this electrode with the two which over-lie the maxillary sinuses, this strip being about 1 inch long by ¼ inch wide, applied over the glabella and down along the bridge of the nose. The plates which fit over the maxillary sinuses are shaped like an oval with a stem extending upward and medially to overlap the small plate that lies along the bridge of the nose. These plates are molded to the maxillary regions so that they fit smoothly, and the stem-like portions are molded down on the bridge of the nose and strapped to the connecting piece which in turn is attached to the frontal electrode with adhesive tape. The electrode clip is then attached to one end of the frontal electrode, the skin underneath being protected from pressure by a bit of heavy rubber, and then a shaped piece of rubber sponge is placed over the frontal electrode and each maxillary electrode.

"The patient holds the rubber sponges over the maxillary plates while the technician wraps an elastic bandage around the forehead to hold the frontal part in place and then around the face to hold the maxillary plates. A plate of about 3x4 inches with well rounded corners is applied to the back of the neck and attached to the other terminal. We usually apply a liberal amount of electrode compound jelly beneath the plates before they are finally bound on.

"The treatment lasts from thirty to forty minutes and the milliamperage must be within the heat tolerance of the patient. Since diathermy margins vary, I cannot give you the exact milliamperage, but with the Victor vario-frequency apparatus this dosage is between 700 and 750 milliamperes.

"Treatments are done daily for the first week, then three times a week. They are always preceded by some shrinkage procedure to insure as good drainage as possible. If there are symptoms of increased temperature, pressure in sinuses, or pain, the treatment is either very much modified as to dosage or discontinued until drainage can be instituted. A rise of temperature is almost proof positive of encapsulated pus in one or more of the treated sinuses. A series of from twelve to twenty diathermy treatments is given and the clinical condition of the patient, of course, determines the question of further treatment."

APPENDIX II

INDEX OF ACADEMY RESEARCH ON SINUS MEMBRANE, 1931-1935

Frontal Sinus Membranes, Cat:

Normal anatomy and histology, vital staining methods 1931

Method of producing acute infection 1931; chronic 1932

Diet, negative effects on progress of infection 1933

Lymphatics 1934; Spread of particulate matter external to membrane 1935

Destructive effects on epithelium: mechanical trauma 1931; heat 1931; paraffin, gum acacia 1931; soap jelly, thin glucose, cocoanut oil, chondrus jelly, milk of magnesia, calcium hydroxide, calcium lactate, scarlet red + oxyquinoline sulphate 1932; typhoid vaccine, merthiolate, alcohol, mercurochrome, dichloramin, trinitrophenol + calcium gluconate 1934; azochloramid, chlorophyll, amniotic fluid 1935

Destructive effects on tunica propria: chondrus jelly 1932; chlorophyll 1935

Negative effects except on cilia: mucilage of tragacanth, petrol-agar, thick glucose, sodium phosphate 1932

Negative effects except increase of goblet cells: dichloramine 1932; histamine 1935

Negative effects on epithelium: roentgen ray 1933, 1935

Negative results: glycin, quartz light, chronic constipation 1935

Acute exacerbation produced by cervical sympathectomy, chronic 1934; acute 1935

Acute exacerbation, chronic membranes: histamine, acetylcholine, chlorophyll 1935

Reparative effects on tunica propria: Mobilization of histiocytes by infection plus trauma 1931; milk of magnesia, calcium hydroxide, calcium lactate 1932

Intradermal vaccination 1932: Does not arrest infective process 1934; peptone, own blood subcutaneously 1934

Round cell infiltration: Destroyed by roentgen ray 1933. Increased by intradermal vaccination 1932; by mercurochrome, metaphen 1934; by ephedrin-resembling drugs, amniotic fluid 1935

Plasma cell infiltration, in most chronic infections 1931-35

Fibrosis: roentgen ray 1933

Prophylaxis against infection: sodium alum, tannic acid 1935

Human Maxillary Sinus Membranes:

Normal histology 1931; differential staining 1932

Reticulo-endothelial system; inflammatory changes 1931

Destructive effects on epithelium: milk of magnesia, normal saline 1935; irradiated oil 1933

Edema of tunica propria caused by sodium and chlorine 1932

Reduction of edema by calcium and magnesium 1932, 1933

Reduction of congestion of mucosa: ephedrin-resembling drugs 1935

Increase of congestion of mucosa: diathermy 1934; acetylcholine 1935

Reparative effects on tunica propria:

- Mobilization of histiocytes: milk of magnesia 1932; calcium gluconate 1933; diathermy 1934; ephedrin-resembling drugs 1935
- Marked plasma cell infiltration: milk of magnesia 1932; calcium salts 1933; diathermy 1934
- Fibrosis: milk of magnesia 1932; calcium salts 1933; roentgen ray 1933, 1934, 1935; diathermy 1934

ALLERGY IN THE RHINOLOGIST'S WAITING ROOM

WARREN T. VAUGHAN, M.D.
BY INVITATION
RICHMOND, VA.

ONE AFTERNOON not long ago, the Havana Special was nearing Savannah, Georgia. On it were three physicians. The train had been blocked and was proceeding slowly. As the three gazed meditatively at the landscape, which for several miles had been monotonously unchanging, one remarked, "This ought to be great hunting country. I'd like to come down here some time with a couple of good bird dogs." The second replied, "I was just looking at all the ragweed along the tracks. Savannah should be a good place for an allergist to settle in." Whereupon the third, coming out of a semi-trance, turned his gaze from the window to the two men opposite him, remarking, "Isn't that a perfectly gorgeous sunset?"

All had been looking at precisely the same thing, but to each the dominant figure in the picture was different. The difference was in the points of view, major interests, or shall I say, in the coloring of the glasses.

The rhinologist and the allergist both look into the same noses, but too often they don't seem to be able to agree as to what they find there. Each has maintained that his own interpretation of the picture was the authentic one. Sometimes one has been right, sometimes the other, but neither has been correct all of the time.

With the ancient Greek physicians there were two schools of belief, that of Cos and the school at Cnidus. So also until recently there were two schools regarding the subject under consideration, schools which disagreed, sometimes bitterly. But history again repeats itself inasmuch as each now begins to recognize the good points of the other and is willing to accept them, nay even to adopt them. Indeed, certain ones of both groups have found that the principle of the old stereopticon is

best, that truest perspective of the picture is attained by fusion of the images observed from both points of view.

Is there any real need for a combined discussion of the subject of allergy by the otolaryngologist, the ophthalmologist and the allergist? Let us allow the former to answer this question. Time allows but a few quotations. L. W. Dean[1] has described the case of a child in which radical operation for pansinusitis was recommended, but in which, fortunately, cytologic studies of the nasal discharge showed an allergic eosinophilia. The child was cured by withholding milk from the diet instead of being submitted to the operation. Dean goes on to remark, "To me it seems obvious that a cytologic examination of the nasal and otologic discharges should be made in every case in which these conditions are present. * * * I should certainly feel that any examination of a nasal case was incomplete if it did not show what cells were present in the nasal discharge."

W. W. Lewis[2] in an essay on *Disillusionments in Nasal Surgery*, remarked, "In my opinion, of all the affections and conditions in the human organism where surgery has been not only the least beneficial, but, in addition, largely harmful, allergic vasomotor and hyperplastic affections of the nose have been the most outstanding. Especially and positively is this so where, in these conditions radical operations upon the sinuses in addition have been done."

Ralph Fenton,[3] in discussing nasal allergy, writes, "Obviously no type of local treatment * * * will do more than afford transitory relief for such people. Adrenalin is a life-saver for the severe case. Ephedrin, helpful at first, becomes an irritant if over-used. Surgery should be limited to the restoration of proper caliber of the nasal passages by thinning down septal deflection; the turbinates should not be slashed out; and, unless bacterial allergy be due to gross infection of the depth of the sinus membrane, the sinuses should merely receive proper aeration and drainage rather than radical measures."

Bernard J. Larkin,[4] in discussing the ophthalmologic aspects of allergy, mentions such conditions as vernal catarrh, uveitis, some forms of conjunctivitis, phlyctenular and dendritic keratitis and certain conditions indistinguishable from trachoma. E. M. Shanklin,[5] in discussing this paper, remarks, "I believe that it is quite true that allergy is being over-worked; too many physicians are exceedingly allergy-conscious." He then adds sev-

eral other eye diseases to the list of possible allergic etiology, namely, retinal edema, various forms of scleritis, blepharitis and follicular conjunctivitis. I would add the very interesting work done sometime ago by Robert H. Courtney[6] on allergy to lens protein seen in some cases of cataract.

Assuming that these and several similar quotations, which time precludes here, indicate that the eye, ear, nose and throat men are today becoming increasingly allergy-conscious, are the allergy cases patiently waiting for recognition in the reception rooms numerous enough or important enough to warrant special effort in this direction? Of the cases of this type that have come to me, I find that far too many have passed unrecognized through the office of the rhinologist.

Before treatment, whether it be rhinologic or allergic, a decision should be reached as to whether the local pathology is due to extrinsic factors, especially inhaled allergen, or to intrinsic factors such as bacterial infection or bacterial allergy. In a series of 139 chronic asthmatics I found 44 to be intrinsic and 95 extrinsic. Of the 95 cases due entirely to extraneous factors, 32, or one-third, had already been subjected to nasal surgery without relief. These cases should not have been operated upon, and would not have been had preliminary allergic studies been made. If the nose and throat man were to recognize all of his allergic material and proceed with appropriate therapy he would do his patients and himself much good. It is usually he who has the first opportunity to reach a correct diagnosis.

How many such patients are sitting, waiting, one cannot say until more surveys have been made, but two very illuminating ones have been reported. Baum[7] has found that among 2,000 routine office cases 10% had allergy referable to the nose and throat and among 700 with primary nasal complaints 27.3% were allergic. Hansel[8] reports that in an analysis of 1,000 routine office cases there were 324 with nasal complaints. Of the latter, 44% had nasal allergy. Roughly, from 25% to 45% of routine cases with primary nasal complaints exhibit an allergic factor. I think we can all agree that the vast majority of cases of vasomotor rhinitis first consult the nose and throat men. Huber and Harsch[9] find 70% of such cases allergic in origin.

In a survey of an entire community of 508 individuals, I have found[10] that approximately 15% have had some upper respiratory symptoms, of mild or severe grade, at some time in their lives, which were attributable to inhalant allergy. The

list of allergenic factors recognized and named by these persons included a wide variety of substances, such as dust, hay, tobacco, smoke, coal gas, pollens, flowers, soap and soap powders, orris root, sunlight, wind, chilling, wheat, flour, feathers, flax, etc.

Now, if it is agreed that all of us here have become "allergy-conscious," and that this phase of medicine is becoming increasingly important in an increasing proportion of nose and throat practice, what should the nose and throat man do about it? Should he learn to pick out the allergic cases, from 10% to 44% of his practice, and forthwith send them to the allergist for study and treatment? I think not.

Fully 50%, possibly more, of cases of nose and throat allergy are relatively simple, sensitive to but a few inhalant allergens which may be readily recognized by appropriate tests, and will be promptly relieved following avoidance or suitable hyposensitization therapy. I believe that, for maximum service to his patients, the nose and throat man should equip himself properly to recognize allergic conditions, to diagnose them by appropriate laboratory and sensitization studies, and to relieve them with proper treatment. There will be a proportion of the cases, from 25% to 50%, in whom the allergy is more complicated or in whom proper allergic therapy requires higher allergen dosages, dosages which carry with them the possibility of anaphylactic reaction unless expertly administered by those who have had long experience in this particular field. These two groups he will be only too glad to send to the allergist for further treatment. I would emphasize here that allergy is not the game for the tyro. He who reads the directions on the commercial diagnostic or treatment sets and proceeds accordingly without the caution of experience will sooner or later get into trouble. Even for routine work such as I am about to outline, the nose and throat man should ground himself in the fundamentals with the aid of an allergist or should have associated with him someone who is well versed in this very special field of medicine.

Assuming that such a grounding has been procured, what allergic procedures could appropriately be carried out by the nose and throat man or the eye man in his own office, with every reasonable guarantee of safety? Time permits but a hasty summary. Fortunately, there will soon be available, in French K. Hansel's excellent monograph on *Allergy of the Nose and Paranasal Sinuses,* a reference volume which will be of utmost value as a guide to the nose and throat man doing this work.

Diagnosis

The first requisite for diagnosis is "allergy-consciousness." One must bear in mind the possibility of allergy as an etiologic factor. Questioning of the patient may substantiate this in the obtaining of a personal or family history of other allergic diseases such as asthma, hay fever, urticaria, migraine, eczema, contact dermatitis or food idiosyncrasy. But even in the absence of all of these the condition may be allergic. One must, therefore, acquaint one's self with the protean manifestations of allergy as they are observed in the eye and in the nose and throat.

As concerns the nose, much information can be obtained by inspection. The pale edematous mucosa of the turbinates and pallor of the septum is most suggestive. Remember that according to Schenck and Kern[11] true mucous polyps are practically 100% allergic.

There is even a rather characteristic allergic facies, as pointed out by Duke,[12] manifested by relatively under-developed malar prominences, due presumably to chronic sinus blockage during the growing period. This produces somewhat of a depression under the eyes or a flattening of the face which is quite characteristic. The V-shaped palate and over-riding upper incisors, commonly called the adenoid facies, has also been designated as an allergic manifestation by Bowen and Balyeat.[13] While I have seen this in allergic individuals, it is certainly not as characteristic as the flattened malar prominences.

Laboratory Diagnosis

Probably the most important diagnostic procedure is one that has been developed by the rhinologist himself in collaboration with allergists. Eyermann[15] first called attention to eosinophilia in allergic nasal secretions. This finding was corroborated by Rudolph and Cohen and the diagnostic procedure was brought to its present reliability by Hansel.[16] The ophthalmologist has a similar procedure available in that Emory Hill[17] showed some years ago that the application of dionine to the conjunctiva elicited a leukocytic response in the conjunctival sac in the smear from which eosinophils could be demonstrated if present.

X-ray of the sinuses is a valuable indirect diagnostic aid especially in identifying mucosal edema and polyps when present. The method of instillation of iodized oil as developed by Proetz[18] is advantageous in that no artificial windows must be made and is occasionally helpful in doubtful cases.

Skin Testing

The commonest inhalant offenders, in my experience, are pollens, house dust, feathers, orris root, pyrethrum, and kapok. While there are many other inhalant allergens of great importance in the individual case, routine tests with the above, will, I believe, enable the rhinologist to diagnose and relieve a reasonably high proportion of his inhalant allergics. The list of pollens used should, of course, include all of the commoner offenders in the locality in which the patient resides.

While the familiar 15 dose treatment set for preseasonal pollen desensitization is remarkably inefficient in my experience, and while the more effective higher concentrations should be given under the supervision of an allergist, the coseasonal method of treatment[19] which has been developed in our clinic is, in my experience, practically free from risk of serious anaphylactic reaction. While its efficiency of 80% is somewhat less than the 85% for proper preseasonal treatment, it so nearly equals it that there would be ample justification for its adoption by the nose and throat man, at least for a season's trial. If found to be non-effective in a particular case, that case can then go on for the next year to allergic supervision with high dosage preseasonal treatment or perennial treatment.

In my own work I always use small dosage therapy in hyposensitization with house dust, feathers and the other allergens mentioned above. This treatment also would, therefore, be safe in the schedule which I am elaborating.

The greatest point of safety that I would emphasize in making the tests is the desirability of preliminary scratch tests, to be followed in case of doubt by intracutaneous testing. Although there have been two or three reported instances of anaphylactic death following scratch test in extremely hypersensitive individuals, they are so rare as to be practically negligible. The intracutaneous test, while usually more accurate, carries with it an increased element of danger, which is minimized by the preliminary scratch test. A positive scratch should not be repeated intracutaneously. The safety element is further enhanced for the physician who has not had wide experience in allergic diagnosis and treatment by the exclusive use of the arms so that an untoward reaction may be controlled by the prompt application of a tourniquet above the site of testing or injection, and the introduction of adrenalin into the opposite arm.

Local Testing

There is a very special field of allergic testing which could well be adopted by the rhinologist. This is the direct application of the test substance within the nose. Certain persons fail to react to the offending substance by skin test, but do react, with the symptoms of the disease, following contact of the allergenic substance with the nasal mucosa — a condition described by Forman[20] as contact allergic coryza.

Diagnostic methods for this condition, first described by Efron,[21] who employed insufflation by means of powder blowers, then improved by Rudolph and Cohen[22] who applied the test substance directly to the turbinates, have been further improved and simplified by Dean.[23] The suspected allergen, in powder form, such as is available commercially for scratch testing, is sprinkled on a wisp of cotton on the end of a toothpick. This is held at the nostril while the patient inhales. In order to preclude too severe a reaction from this procedure it is better to reserve it only for questionable cases in which the cutaneous reaction has been found negative. Otherwise, only extremely small quantities should be used very cautiously. Dean also applies the direct nasal test by intramucosal injection.

Discussion

The above is certainly a minimal outline of allergic procedure for the nose and throat man. He should have in his office the equipment for microscopic examination of secretions and for the few routine tests as I have outlined them. He could do more, such as, for example, the food tests. But since these require a wide experience in interpretation, and correlated studies such as the leucopenic index and the food diary, they are likely often to become more confusing than illuminating for the man whose interest is only secondarily in allergy. The appropriate technic for the methods as outlined above is available in the current literature. The point which I would emphasize here is that if this minimal program of inhalant allergic study fails to relieve the patient, one should not conclude that allergy is not responsible for the symptoms. There is much more to complete allergic studies than what I have outlined. If one is reasonably sure in a given case that allergy is playing a part or if, in spite of positive reactions, the patient fails to respond, then, in justice to the patient he should be sent on for more detailed allergic study. Without having actually put it to the trial in

the manner described, I will venture a guess that the rhinologist who carries out even this minimal program will find it well worth his time and effort in a sufficiently high proportion of those patients who otherwise would eventually become discouraged and, on their own initiative, consult an allergist. I know of at least one otolaryngologist who has gone much farther. Even though he had had no previous practical experience in allergy, he some time ago procured a competently trained allergy technician who under his supervision has carried the studies into as great detail as is done in the offices of many allergists. His results have been satisfactory in every way.

I have not mentioned bronchoscopy in asthma, in which, occasionally, the laryngologist is of greatest aid to the allergist in removing adherent secretions or other obstruction, and in ruling out malignancy or other non-allergic pathology.

Conclusions

May I conclude this discussion with the plea that the eye, ear, nose and throat man realize that there are undoubtedly several unrecognized patients with allergic disease now sitting in his waiting room or patiently submitting to repeated local treatment in the hope of ultimate relief; and with the plea that he utilize those procedures which are already available and which will enable him to give more speedy relief to a surprisingly large proportion of his patients.

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DISCUSSION

Dr. French K. Hansel, St. Louis: Dr. Vaughan has pointed out the general incidence of allergy among the population at large and especially in the practice of otolaryngology. In a general way, he has outlined the methods of clinical and laboratory investigation.

In order to give you some idea of the nature of the cases of primary nasal allergy seen in otolaryngologic practice, may I present briefly some statistical data obtained from an analysis of 420 cases in adults and children. It is especially noteworthy that only a small percentage of patients with the nasal manifestations of allergy do not have other types of respiratory allergy or other manifestations, such as headache, gastrointestinal symptoms and skin lesions. Among 220 adult patients, 28% had only nasal symptoms; the remaining 72% had hay fever, asthma and other manifestations of the skin, gastrointestinal and headache types. Approximately 25% of the patients had hay fever; 20% had asthma; and taking the entire group as a whole, approximately 70% had other allergy besides the respiratory type. It is evident, therefore, that it is the rule for patients with nasal allergy also to exhibit other manifestations of this disease.

Among 200 children, somewhat similar percentages were noted. About 25% of the children had hay fever. It is especially noteworthy that 70% of the children had asthma and that only 25% had nasal symptoms without hay fever or asthma. Taking the group as a whole, 68% of the children had other manifestations of allergy besides the respiratory type.

Considering both adults and children, about 25% have hay fever, but we have found that approximately 75% of all patients with hay fever also have active nasal symptoms outside the hay fever season. Only about 25% of the patients with hay fever, therefore, have purely seasonal symptoms. As in the case of nasal allergy in general, about 70% of the patients with hay fever have associated manifestations of allergy of the skin, headache and gastrointestinal types, and, therefore, other sensitivities besides pollen.

In the routine investigation of the nose and paranasal sinuses in cases of gastrointestinal disease and headache, it is especially important to determine whether the nasal manifestations of allergy exist, for their presence may offer good presumptive evidence that these other symptoms are of an allergic nature.

In general, these statistics show that, as a rule, the nasal symptoms are frequently accompanied by other manifestations of allergy and indicate the importance of treating these patients primarily from the allergic standpoint. The use of non-specific local and general methods of treatment should be reserved for those patients who have not responded to recognized allergic methods.

In the diagnosis, especially of the atypical cases of nasal allergy, and in order to determine more accurately the presence of acute or chronic infection, routine cytologic studies of the nasal secretion should be made on all nasal and sinus cases.

In Dr. Vaughan's conclusions, I wish he would speak just a few minutes in telling you something about his work on allergic headaches, especially what he found in the investigation of true migraine, and of the allergic type of headache.

Dr. Hugh S. Kuhn, Hammond, Ind.: I have always had a great interest in knowing just who sits in the waiting room and also how to encourage an increasing number of patients to sit in my waiting room. It is very refreshing to be told by Dr. Vaughan, with very carefully outlined procedures, how we can increase our efficiency and effectiveness as rhinologists.

Some time ago I asked Dr. Vaughan what an ordinary rhinologist should do about cases which he suspected of having an allergic background. He replied that the least one could do was not to operate on them until he or someone else had answered the allergic question, and he then helped me organize my office and secure a competent technician, Miss Blanche Ullman, from his own staff, to carry on these studies.

For the past two years we have attempted to rule the allergic factor out as often as we suspected it. I am sure that the allergy that sits in

our waiting room has been turned into a much more remunerative and a far less worrisome group than formerly. We find some that we can completely relieve by allergic management; others relieved by allergic management combined with surgical and medical care; and the pure allergics that must first go to an allergic specialist before we dare do any surgical procedures in the nose. I have been amazed at the clearing-up of noses blocked full with polypi by taking sleeping pillows and a list of foods from the environment.

The title of Dr. Vaughan's excellent paper stimulated my curiosity as to my own practice and I dove into the active files to see just what was passing through our hands. The following tables show an analysis of proportions of types of cases and the allergic volume which comes to an eye, ear, nose and throat office for relief.

TABLE I — A Review of 3657 Consecutive Cases in an Eye, Ear, Nose and Throat Practice

A. Number coming with eye complaints	1665	45%
B. Number coming with ear complaints	692	19%
C. Number coming with throat complaints	580	16%
D. Number coming with nasal complaints	720	20%

TABLE II — A Review of the 720 Nasal Cases Shows:

A. Allergic nasal cases	140	19.4%
B. Other than allergic nasal cases	580	80.6%
C. The allergic nasal cases are of total cases reviewed		3.7%

TABLE III — Review of the Allergic Cases

A. Showing an eosinophilia
   1. Over 50% ..... 20 cases
   2. 10% to 50% ..... 34 cases
B. Showing no eosinophilia (1-10%) but proven allergic ..... 28 cases
C. No smears taken ..... 52 cases

TABLE IV — Review of the Allergens to Which These Allergic Nasal Cases Were Skin Sensitive:

A. Inhalants	57	40.7%
B. Foods and Contacts	7	5.0%
C. Both Inhalants and Foods and Contacts	76	54.3%

TABLE V — Review of Results

A. Satisfactory by allergic management alone (avoidance and desensitization)	67	47.0%
B. Satisfactory by allergic management plus local treatment	38	27.0%
C. Satisfactory by allergic management plus surgical management	30	21.7%
D. Failures	5	3.5%

Dr. Herman Semenov, Los Angeles: As rhinologists, we see the pathological anatomy of allergy, particularly the degenerative changes within the nose and sinuses; and since allergy is a confusing subject, even the allergists differ among themselves on definitions and treatment, I sometimes believe that the subject is in danger of becoming top-heavy. In this connection, I am inclined to differ with those who assert that all nasal polypi (100 per cent) are allergic.

Polypi are sometimes allergic and sometimes inflammatory, being manifestations of constant congestion and edema, regardless of cause, in a delicate stroma, as the following slides show.

Here we have the submucosa of the sinuses showing a loose layer of areolar stroma. The areolar tissue spaces breakdown when any great infiltration of fluid occurs, as for example the edema in allergy.

This specimen is allergic. The areolar tissue is very edematous and the hydrostatic pressure of the fluid is disrupting the integrity of the entire membrane.

The next specimen is not allergic; it is a clean-cut case of chronic inflammation, pyogenic sinusitis. Here are exactly the same polypoid folds.

When inflammatory edema subsides it leaves behind it a certain amount of tissue hyperplasia. The interesting thing about allergy is that it tends to be diffuse and often involves all of the membrane in all of the sinuses.

On the other hand, when the edema persists for a long period of time —off and on for ten or fifteen years—the end result is polypoid and cystic degeneration.

Cysts appear in weakened connective tissue. This specimen shows two different kinds of cysts. The retention cysts contain mucus, the connective tissue cysts are devoid of epithelium and contain serum. Large cysts are generally accompanied by microscopic cysts. For this reason one should remove the entire membrane and leave a clean cavity. The next slide shows postoperative healing. The cavity is lined by a firm layer of scar tissue, less tendency to become cystic, and the surface may or may not be covered over by a layer of ciliated epithelium.

There are some mucous membranes which are more or less fibrous to begin with and these tend to heal spontaneously. In this slide we have such a specimen. The epithelium is perfectly normal after twenty-five years of sinusitis.

In an allergic membrane there is much degeneration of the epithelium and large areas are denuded altogether. The membrane is open to infection and since the cilia are lost has no way of draining the cavity. Incidentally, there is no reason why an allergic individual may not have an empyema of the antrum superimposed on the allergic condition, for example, an extension of infection from a tooth entering the antrum. It is

unfortunate that bacterial allergy was not discussed by the speaker in this connection.

I wish to mention, before I close my discussion, that I have just examined 500 temporal bones in the Otological Laboratory at Johns Hopkins and have not seen any histological signs of allergy in the middle ear.

DR. WARREN T. VAUGHAN, closing: I don't know whether any of you have ever attended a meeting of allergists, but if you haven't, you have seen the beginning of one now. There is plenty of difference of opinion, and that is what makes the subject so interesting.

About bacterial allergy, I don't know that there are so many who accept the specificity of clinical bacterial allergy. We would all love to do it because all of the early work on experimental anaphylaxis points to the logical conclusion that there is such a thing as a specific bacterial allergy, and yet with the methods that we have had available up to the present time any attempt to show that bacterial allergy is a consistently active specific process has been a failure much more often than it has been successful.

I believe that there must be some specificity to bacterial allergy, but when it comes to trying to make something out of it in the specific treatment of intrinsic allergic diseases, of those that are supposed to be due to bacterial allergy, we get our poorest results. With our extrinsic cases, in my own series, we get, even in perennial asthmatics or perennial vasomotor rhinitis, 68 to 70% satisfactory results, while with perennial intrinsic vasomotor rhinitis or asthma, we get the poorest results, around 30 to 40%. I can assure you gentlemen that in our work, as well as the work of others interested in allergy, we have done our best to show a specific bacterial factor. We do use vaccines. We use vaccines a great deal in my own office, but we use them usually with a realization that they are probably not specific. I say "probably" because we don't know where we are on the subject of bacterial allergy. All I can say is that up to the present there are only three men whom I know doing allergic work who insist on the specificity of bacterial allergy. All of the rest feel that we aren't getting anywhere.

Now it is entirely possible that it is the technic, or the preparation of the vaccine, or something like that, and that someone will get there sometime. I admire any man who starts to dig into bacterial allergy, because he has a lot of work to do.

I do agree with Dr. Semenov in his statement that it is the rhinologist who has to work out the subject of the allergic factors in his own field. That is absolutely true.

The rhinologist, together probably with the pathologist, possibly has put a little too much stress on the interpretation of polyps, whether they are allergic or not, by the presence or absence of eosinophils. You can't always determine whether a polyp is allergic or not allergic by the presence of eosinophilia because that is a temporary affair.

I quoted Kern and Schenck, in making my statement about 100% of polyps, and in their defense, I must reiterate that they didn't say 100% of polyps are allergic; they said 100% of mucous polyps are probably allergic. Many of you, no doubt, heard Schenck's paper at the meeting of the American Medical Association in Cleveland. I won't take the time to detail their reasons for reaching this conclusion.

While the subject of migraine is a tremendously interesting field, I think I should not take the time to discuss it here, and if Dr. Hansel will excuse me, I won't get started on that.

THE EFFECT OF RADIATION UPON CILIATED EPITHELIUM

LYMAN H. HEINE, M.D.*
BY INVITATION
BOSTON

* * *

INTRODUCTION

VARIOUS THEORIES have been advanced as to the normal physiology of ciliated epithelium and how the behavior of the cells forming this type of epithelium has been altered by drugs, temperature, and the surrounding atmosphere.

Hilding's earliest work uncovered a rich field for research and medical literature has been filled with reports upon all phases of this interesting problem since he published his findings.

Cilia — delicate, weak, frail cilia — such is the popular conception of the type of epithelium on which we have placed a large amount of responsibility for the protection of our nasal cavities from disease. That nature is more kind than to place a really delicate structure in direct fire of the enemy has been lost sight of by all but a few. Proetz, however, early in his experiments, was impressed by the hardiness of so delicate an appearing cell. It is hoped that, at the conclusion of this paper, all misgivings as to the delicacy of this structure will have been lost, especially when it is realized that single massive dosages of roentgen ray as high as 24 erythemas† permit these cells to live.

It was at Dr. Mosher's suggestion that a study be made to determine the effect of radium upon tissue cultures of ciliated epithelium. The dosage was to be sufficient to kill the cells at various points in their life cycle, and our objective was to learn something of their behavior. A number of problems arose and one or two interesting facts revealed themselves. These will be dealt with at the conclusion of this paper.

* From the Mosher Laboratory, Massachusetts Eye and Ear Infirmary.
† Wherever erythema is used, "skin erythema dose" is implied.

Observations Upon Living Ciliated Epithelium Following Exposure to Radium and X-ray

As one pursues a course of investigation, in some particular field, the original line of thought is often interrupted and some other problems are discovered, while one is attempting to carry out and prove the original idea.

Our purpose in this work was to determine the action of radium upon tissues cultures. However, in the light of subsequent developments, the word "cultures" has been dropped and the words "living ciliated epithelium" used. In other words, the following observations are made upon living cells, and not the growth of these cells.

Method — Our observations were upon three different types of experiments, each of which will be mentioned and methods used in each will be outlined.

Dr. Mosher suggested the use of tracheal tissue from rabbits, since it is always available. This organ furnished most of the ciliated epithelium, except in one case, and in that experiment the cilia were active for 8 days. In this experiment the epithelium was removed from a nasal polyp. Though rigid adherence to sterile technique was not in force, all instruments, glassware, and solutions were sterilized.

Pieces of tracheal tissue, 1-2 mm. in size were removed and placed in a hanging drop. One mount was used as a control and one was exposed to radium. The tissue was incubated at 28° C. Radon seeds of 1 μ each were used. These were placed over the cover glass in a circular manner and were never any more than .5 cm. away from the tissue. The cover glass did not contain lead. The seeds were left in place until the death of the tissue.

The following observations were made:

Shortly after placing 6 μ seeds about the tissue, ciliary motion increased, and there seemed to be an increase in ciliary activity with the placing of every dose.

Thirteen attempts were tried at varying the total dosage. However, it soon became apparent that the results were very inconsistent. About the only observation worth while that was made was the little effect of radiation on living ciliated epithelium. It was then thought that by using X-ray the following factors could be controlled.

(1) The dosage could be accurate.
(2) The tissue could be exposed directly to the action of the ray.
(3) Very high dosage could be obtained.

The following plan of using X-ray was adopted.

After removal of the trachea from the rabbit it was cut into equal portions and each portion washed thoroughly. One specimen was placed in a test tube containing Ringer's solution. The other was laid upon a piece of sterile gauze and kept moist by means of slowly dropping Ringer's solution. This specimen was exposed to the roentgen ray. The rays were not filtered and the target skin distance was eight inches. In all cases, single exposures were employed, the machine running at 100 kv. and 5 milliamperes.

Following radiation, a small piece of the mucous membrane was removed and placed in the hanging drop. The rest of the tissue was placed in a test tube containing Ringer's solution and kept at a temperature of 14° C. The same was done with the control. From time to time as the hanging drop specimens died, new specimens were secured from the tissue kept at 14° C.

Dosages were started at 4 erythemas, and will be reported in the order of experimentation.

The tissue receiving 4 erythemas lived 120 hours, the control dying at the same time. No visible effects on the ciliated cells or upon the activity of the cilia were noted.

Eight erythemas (2800 r.)
The tissue lived 168 hours
The control lived only 48 hours

Sixteen erythemas (5600 r.)
The tissue lived 168 hours
The control lived 144 hours

Twenty-four erythemas (8400 r.)
Specimens receiving X-ray lived 314 hours
The control lived but 216 hours

After 120 hours, the irradiated tissue seemed a little more active and the amount of débris in the surrounding medium was less than that of the control. By débris is meant dead cells, for the most part, and leukocytes.

At this point the question arose as to whether or not the metabolism of these cells was being affected. It was decided that some idea of the metabolic rate might be obtained by determining the p_H of the solutions containing the tissue.

Therefore, dosages were started at 16 erythemas, or 5600 r., and increased to 32 erythemas, or 11,200 r.

The p_H was determined upon the fresh Locke-Ringer's solution, upon solutions containing the irradiated tissue, and solutions containing control tissue. Since some CO_2 might be absorbed from the air, p_H determinations were also run upon solutions of Locke-Ringer's that were exposed to the same experimental conditions as the other solutions.

In general, the solutions containing the irradiated tissue and the control had a p_H of 6.0, the fresh Locke-Ringer's solution of 7.3, and the control solutions of 6.4.

It would seem the variation between the control specimens and the irradiated tissues was not enough for any conclusion as to acceleration or retardation of metabolism.

The viability of the tissues subjected to the increased dosage of 32 erythemas is of considerable interest. The three specimens exposed to this dosage lived 48, 96, and 48 hours, respectively. The controls lived 96, 168, and 72 hours.

From these results one concludes that an X-ray dosage of 11,200 r. is required before any gross effect upon the viability of the ciliated epithelium is detected. For the first time during the course of the experiments the control tissue lived longer than the irradiated tissue. In this respect it is of interest to quote the following from Dr. Fenton's paper. "Study of tissues from infected antrum mucosa and cyst walls from a man of thirty-six who had received doses of deep roentgen rays (because of suspected malignancy) showed enormous thickening and fibrosis of the tunica propria, many plasma cells, and almost normal epithelium."

The third portion of this paper deals with the actual implantation of radon seeds in the nostrils of rabbits. The procedure was carried out along the plan suggested by Dr. Mosher. The same number of seeds was used in each case, the variable being the length of time the animal was allowed to live following exposure.

Five 1 μ seeds were used. The seeds were partially imbedded in a small mould of dental compound and then inserted in the nostril. The mould was then secured by means of a suture partially closing the nostril. It was hoped that in this manner a rather superficial effect might be secured and that the length of time the nostril was closed would not be sufficient to produce the changes noted by Hilding. At the end of three days the sutures were removed. In some cases they had sloughed out. The animal was allowed to live four days after radium implantation.

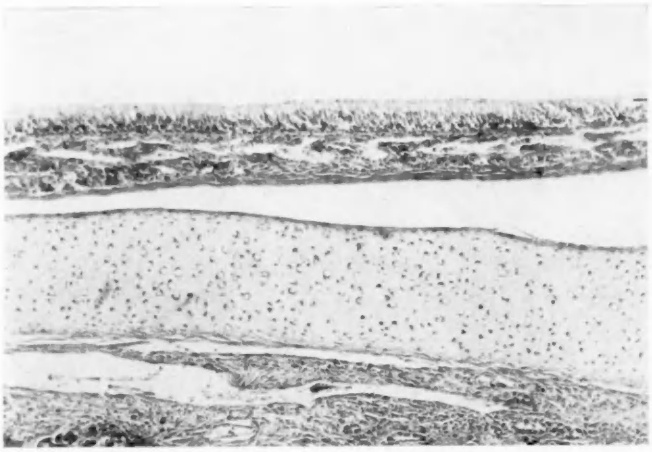

FIG. 1 — (Heine) — This section is of the left side of the septum, opposite the side of radium implantation. The epithelium is essentially normal. The cell outlines are distinct. The nuclei are normal in appearance. The cytoplasm is clear. There are a few goblet cells but these are not excessive. *A*, Epithelium.

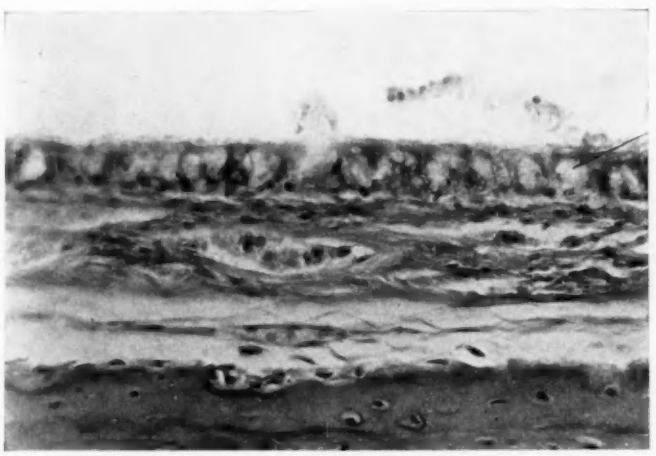

FIG. 2 — (Heine) — This section from the same animal is from the right side of the septum. The animal was allowed to live four days following radium implantation. Note the large number of goblet cells. The cell membrane is no longer distinct, the nuclei are pycnotic, and are very indistinct, whereas the cytoplasm of the cell is a little more glandular. *A*, goblet cells.

FIG. 3 — (Heine) — This section is from the same rabbit and the side exposed to radiation, but was not as near to the radium in that it is from the inferior turbinate. Notice the very high type of epithelium and, judging from the number of cells, it would appear as if the epithelium is hyperplastic. *A*, Epithelum.

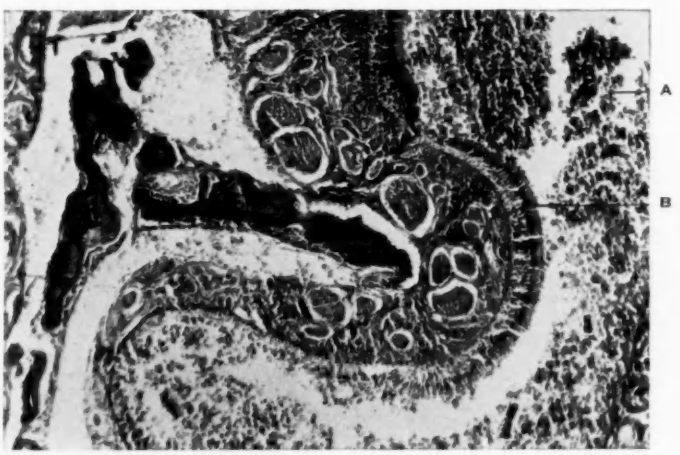

FIG. 4 — (Heine) — This section corresponds to figure 3, but is from the left side, the side without radium. The epithelium is hyperplastic in this case. However, considerable evidence of infection is present in this specimen, and it may be a hyperplasia for that reason. *A*, Inflammatory exudate; plastic. *A*, Epithelium.

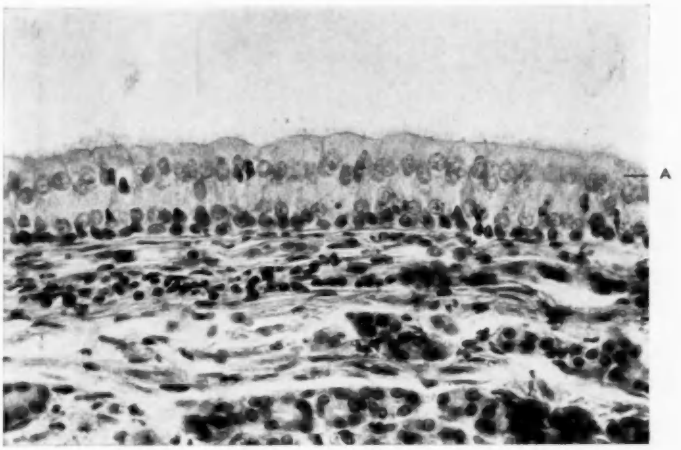

FIG. 5 — (Heine) — This is from the unradiated side of the second rabbit living fifteen days following radium implantation. It is a beautiful example of normal ciliated epithelium from the septum. Cell outlines are distinct, the nuclei are normal, and no hyperplastic changes are noted. *A*, Epithelium.

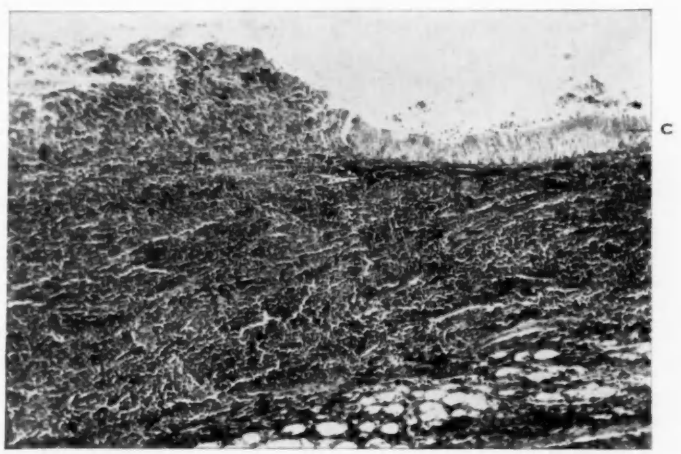

FIG. 6 — (Heine) — This is from the radiated side of the rabbit. In this section there is a hyperplasia of the epithelium about an area where the epithelium seems to have been destroyed. Close observation reveals many epithelioid cells, but no order of arrangement and no cellular outline. There is a dense infiltration of lymphocytes. *A*, Epithelioid cells; *B*, Infiltrated area; *C*, Epithelium.

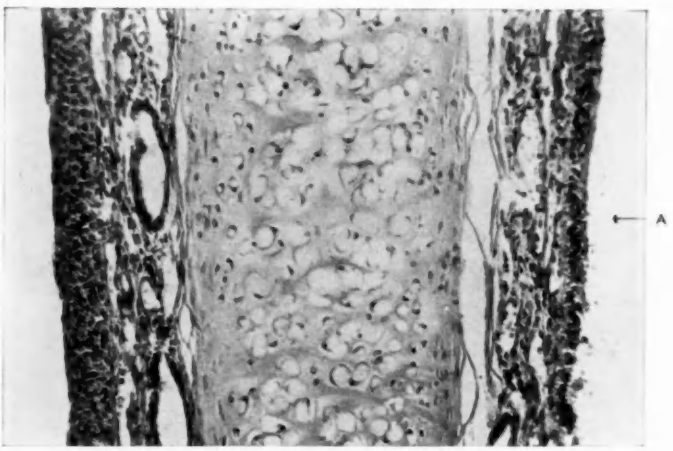

FIG. 7 — (Heine) — This section shows both sides of the septum. The animal was allowed to live two months following irradiation. The side opposite the radium shows stratified squamous epithelium, whereas the side of irradiation shows destruction of the epithelium down to the basal layer. *A*, Irradiated side.

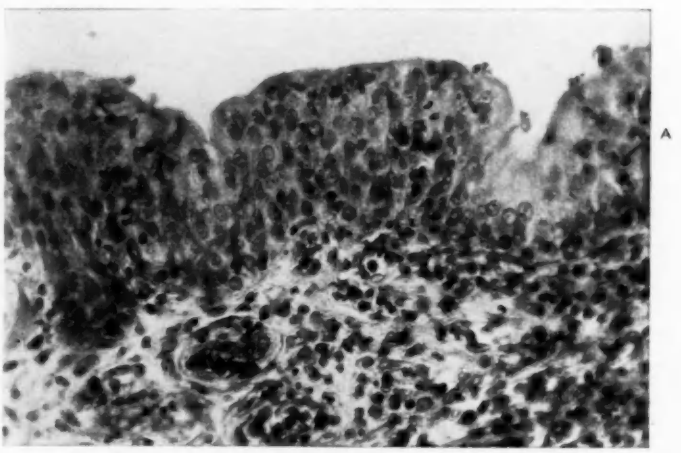

FIG. 8 — (Heine) — High power of section of rabbit's nasal mucosa removed from the septum two months following irradiation. The epithelium is markedly hyperplastic. Some degree of its activity may be obtained by the number of mitotic figures present. *A*, Mitotic figure.

Section I — This section is from the left side of the septum, opposite the side of radium implantation.

The epithelium is essentially normal. The cell outlines are distinct. The nuclei are normal in appearance. The cytoplasm is clear. There are a few goblet cells, but these are not excessive.

Section II — This section from the same animal is from the right side of the septum. Note the large number of goblet cells. The cell membrane is no longer distinct, the nuclei are pycnotic, and are very indistinct, whereas the cytoplasm of the cell is a little more granular. This section illustrates what I believe to be the early changes resulting from radiation.

Section III — This section is from the same rabbit and the side exposed to radiation, but was not as near to the radium in that it is from the inferior turbinate. Notice the very high type of epithelium and, judging from the number of cells, the epithelium appears to be hyperplastic. If so, this type of epithelium is similar to that described by Latta and Schall as being hyperplastic as a means of furthering protection.

Section IV — This section corresponds to section III, but is from the left side, the side without radium. The epithelium is hyperplastic in this case. However, considerable evidence of infection is present, and it may be a hyperplasia for that reason. It is unfortunate that we can not say that radium produces epithelial hyperplasia here as it did on the opposite side.

Section V — This is from the unradiated side of the second rabbit, living 15 days following radium implantation. It is a beautiful example of normal ciliated epithelium from the septum. Cell outlines are distinct, the nuclei are normal, and no hyperplastic changes are noted.

Section VI — This is from the irradiated nostril of the rabbit. In this section there is a hyperplasia of the epithelium about an area where the epithelium seems to have been destroyed. Close observation reveals many epithelioid cells, but no order of arrangement and no cellular outline. There is a dense infiltration of lymphocytes.

Section VII — This section shows both sides of the septum. The animal was allowed to live two months following irradiation. The side opposite the radium shows stratified squamous epithelium, whereas the side of irradiation shows destruction of the

epithelium down to the basal layer. It is quite probable that the radium has made its effect manifest upon the side showing the stratified epithelium, as well as the side devoid of epithelium. Consequently, that side has undergone hyperplastic changes to the point of an absolute change in morphology, whereas the side receiving the full dose of radium has passed through this stage and has entered the stage of destruction.

Section VIII — This is an illustration of the hyperplastic changes mentioned above. Section from another animal living two months following radiation. Notice the mitotic activity.

From these studies one concludes that ciliated epithelium undergoes the following changes following radiation.

1. Hyperplasia.
2. Loss of cell outline and the production of pycnotic nuclei.
3. Goblet cell formation.
4. Destruction down to the basal layer.

The minute cellular changes produced by X-ray have been thoroughly studied and published in a volume by Colwell. These studies are concerned with types of tissue other than ciliated epithelium.

For the sake of completeness, we are attempting studies upon the mitochondria and Golgi network in ciliated epithelium following exposure to X-ray.

The functions of these two cytoplasmic inclusions are still not definitely known. Many workers, however, contend that the mitochondria have to do with respiration. Therefore, the possibilities of research along this line are rather exciting.

Summary

The foregoing paper has recorded observations made upon the behavior of ciliated epithelium after exposure to radiation. Three methods of approach to this problem were used: first, the effect on hanging drop specimens of ciliated epithelium by exposing them to the action of radon seeds; second, the results obtained by exposing sections of rabbit's trachea to the direct action of the roentgen ray; third, the after-results from implantation of a fixed dosage of radium in the nostrils of rabbits, determined by observations made upon the histological specimens at varying lengths of time after the implantation.

Conclusions

1. Goblet cells may be another form of ciliated epithelial cells. Furthermore, they are probably degenerative as shown by changes noted in the nasal mucosa of rabbits after exposure to radium.

2. The metabolic rate of the cells evidently was not affected by roentgen ray when this metabolic rate was measured by changes in the p_H of the solutions containing the tissues.

3. It may be possible that some dosages of roentgen ray are actually stimulating. Therefore, it should be determined what dosage will produce beneficial results.

4. Ciliated epithelium is not a delicate structure. If one considers that a single dose of unfiltered roentgen ray of 8 erythemas is the maximum for the skin of man, the picture of the severeness of a burn produced by a single dose three times that amount, or 24 erythemas (skin erythema doses), is almost beyond one's imagination. However, these experiments indicate that these frail-appearing cells do stand such a dosage with impunity.

In searching for an answer to the question, "Why are these cells so remarkably viable?" — I can find no better explanation than the one offered by Dr. Werner Mueller; namely, that cilia are an ancient structure biologically, and, therefore, in the order of events, they should be the last to be destroyed.

DISCUSSION

Dr. H. P. Mosher, Boston: It was very nice of you to receive this paper so kindly. The abstract was thorough and so much to the point that there is very little for me to add.

The main point of Dr. Heine's paper is that cilia are astonishingly resilient to the action of X-ray. I, for one, had looked upon the cilia as extremely fragile. I think that has been the general view. However, as far as their reaction to X-ray goes, they are anything but fragile.

The second point brought out by Dr. Heine is the increase of goblet cells after the exposure of the animal to radium. He feels that this is in line with the theory that goblet cells are degenerative cells.

In carrying out the experiments we were so startled by the lack of results, that the question came up whether or not there was a "nigger in the woodpile" somewhere; in other words, was our X-ray machine delivering the dosage it was supposed to deliver? Dr. Holmes, the head of our X-ray Department, shared this doubt, but was very nice about it. The machine was checked up and it was found that it was giving the dosage it was supposed to give. Without the cooperation of our X-ray Department

Note: The author wishes to express his sincere thanks to Dr. G. W. Holmes and his staff of the X-ray Department at the Massachusetts General Hospital for the use of the X-ray equipment and radon seeds necessary for the carrying out of these experiments; likewise, for such technical advice as was essential for their use. The photomicrographs were taken by Dr. Werner Mueller.

this investigation, of course, would have been impossible. It goes without saying that a large amount of work was put into the investigation.

DR. RALPH FENTON, Portland, Ore.: I am tremendously impressed with the magnificent results of the hard work that Dr. Mosher gets out of his young men, and it should be an inspiration to those others in the United States who are teaching residents and associates to see just what Dr. Heine has been able to do with this very difficult job.

This has reduced to an absolutely positive set of facts, the sort of thing which we attempted to do a few years ago in the course of our Academy research, when we tried to check up on some people who claimed they were curing sinusitis by the use of the X-ray. We made some cats thoroughly sick for them, and they pitched in and irradiated the cats and found out on a series of animals, from 24 hours to 6 weeks after irradiation, the only result that was caused, on top of the chronic infection which was already present, was that the epithelium remained unchanged, except at the very first when the changes may have been due to a residual chronic infection.

There was, however, a factor which I think is not to be considered in these beautiful specimens of Dr. Heine's, one factor in these infected membranes and in human membranes which we examined in the same series which does not exist here, namely, that the X-ray destroys lymphocytes. The dead bodies of those lymphocytes have to be carried away some place. Also the immune substances, seeping out from the dead bodies of the lymphocytes in the tissue, go places and bring about the inrush of tissue phagocytes and other types of reparative cells and eventually lead to the fibrosis which Dr. Heine mentioned and which I had spoken about earlier this afternoon. That fibrosis is the thing which the X-ray man can bring about in his attempted treatment, but he may entrap — just as you may entrap in a sore tonsil, part of which is chopped off or part of which is shriveled up by the X-ray — disease germs in such a fibrous membrane deep down in the stroma. Neither the X-ray nor radium has any antiseptic effect whatsoever. The preliminary irritation of the epithelium pointed out by Dr. Heine may possibly be due to the exudation through the membrane outward, between the epithelial cells of some of these broken-down cell substances from cells that are destroyed in the tunica propria.

DR. SAMUEL IGLAUER, Cincinnati: The chair would like to ask a question — that is, Did he rule out the effects of infection when radium was implanted in the nose of the rabbit? Because the nose of the rabbit is very easily infected by any manipulation.

DR. LYMAN H. HEINE, closing: In answer to the question, it was very difficult to rule out infection. The only thing that we had to go by was a series of normal slides and comparisons and by study of the changes that were produced by infection, as you know it to occur. In this one slide that I have shown we show the infection that was undoubtedly infection, and on the opposite side it was believed to be clear of infection.

I wish to thank Dr. Mosher and Dr. Fenton for the discussion of the paper, and also the members of the Society who have accepted it so kindly.

MALIGNANT TUMORS OF THE EPIPHARYNX

SAMUEL SALINGER, M.D., AND
SAMUEL J. PEARLMAN, M.D.

CHICAGO

IN PRESENTING this study of 24 cases of malignant tumors of the nasopharynx, we wish to emphasize particularly the histopathology and if possible to clarify the nomenclature of a type of growth which we believe is characteristic of this location. The literature is full of confusions and contradictions in the designation of these growths so that it is often difficult to understand what is meant by the terms various authors have employed. We have examined most of the reports available on this subject and find the cases, roughly speaking, fall within three groups, namely, Carcinomas, Sarcomas, and Endotheliomas. The subdivisions of these groups are numerous and add much to the confusion of terminology. For instance, under Carcinomas we find the following types: squamous, scirrus, non-epidermoid or baso-cellularis, epidermoid or spino-cellularis, intermediary epidermoid, anaplastic, adeno, undifferentiated, transitional cell carcinoma, and lympho-epithelioma. Under Sarcomas we find lympho, spindle cell, round cell, myxo, fibro, chondro, reticulum and polyhedral cell sarcoma. Endotheliomas are a group by themselves, the frequency of which has been stoutly supported by some and as vigorously denied by others. In addition to the wide range of terms designating these tumors we found that where large series of cases were reported there was a further discrepancy in the distribution into the two major groups (Carcinoma and Sarcoma) which ran counter to our own observation. For instance, New[1] reported 194 cases of which 154 (80%) were highly malignant epitheliomas and 40 (20%) lympho-sarcomas. Harmer[2] collected 365 cases of which 86 (24%) were carcinomas, 232 (63%) sarcomas and 47 (13%) endotheliomas. Zuppinger[3] with 32 cases had 15 (47%) carcinomas, 15 (47%) sarcomas and 2 (6%) alveolar type. Crowe and Baylor[4] reported 40 cases, 26 (65%) being sarcomas and 16 (35%) carcinomas. Oppikofer[5] 21 cases,

6 (30%) carcinomas, 11 (52%) sarcomas and 3 (18%) endotheliomas. Citelli[6] 10 cases, 4 (40%) sarcomas, 5 (50%) carcinomas and 1 (10%) endothelioma. Ewing[7] with 110 cases found 47 (43%) transitional cell carcinomas, 30 (27%) squamous cell carcinomas, 11 (10%) lympho-epitheliomas, 15 (13%) lympho-sarcomas, 4 (4%) adeno-cystic epitheliomas and 3 (3%) malignant adenomas. Heine[8] reports 19 cases, 13 carcinomas and 6 sarcomas. In addition to these we have found at least fifty other authors reporting from one to ten cases in which there was an astonishing lack of agreement as to nomenclature or histologic classification. These facts are mentioned because they are in marked contrast to our own findings in 24 cases including 5 with complete autopsies. The slides from all of these cases were independently examined by three pathologists and their diagnoses compared. A glance at the accompanying table discloses a remarkable agreement of opinion. If we included epidermoid carcinoma, undifferentiated squamous cell, anaplastic and transitional cell carcinoma within one group because of their similar origin and common characteristics we find that the great majority of our cases come within this category. Furthermore, we found that both sarcoma and endothelioma were extremely rare as our pathologists could agree on but one of the former (two others being doubtful) and the one case which was named endothelioma was called adenoid-cystic carcinoma and basal cell carcinoma with invasion of blood vessels by the others. Considering the fact that the large majority of our cases were definitely placed in the classification of anaplastic epithelial growths (we believe most of these are transitional cell carcinomas) it is difficult for us to reconcile these findings with those of the authors we have mentioned. We can only assume that the lack of agreement is due to diverging conceptions of the histopathology in these cases because it is inconceivable that a series of 24 cases should run so completely into one definite type of growth. We are convinced that aside from the Broders classification which is based on the percentage of immature cells without regard for their type or arrangement, most of the other terminology has been in error and that many cases heretofore termed "sarcoma" or "endothelioma" were in reality transitional cell carcinoma or lympho-epithelioma.

Since Schmincke[9] and Regaud[10] first called attention to the characteristics of lympho-epithelioma by which they were to be differentiated from other malignant tumors and since Quick and

Cutler[11] first adopted the term transitional cell carcinoma as a histopathologic entity, we have been able to trace in the literature a more and more definite tendency to recognize and describe them and a disposition to drop many of the other terms by which these tumors were designated. For instance, we find less mention of endothelioma in the more recent literature and are firmly convinced that it is an extremely rare growth. We feel quite sure that many of the cases reported as such by Trotter,[12] Gardham,[13] Zschuncke,[14] Oppikofer,[5] Saareste,[15] Citelli,[6] Aisendorf[16] and others were in reality transitional cell carcinoma or lympho-epithelioma. Our opinion is based on a comparison of such descriptions or microphotographs as these authors have published with the sections from our own series as well as those of Quick,[11] Cutler,[17] Cappell[18] and others. Cappell, in a particularly illuminating article replete with microphotographs, has shown varying types of lympho-epithelioma and transitional cell carcinoma of the tonsil and nasopharynx and has pointed out the essential characteristics by which the diagnosis is made. The tubular and alveolar arrangement of the cells by which many authors recognized endothelioma were shown to coincide with the classical descriptions of lympho-epithelioma as given by Schmincke[9] and Jovin.[19] Also the resemblance to Sarcoma was pointed out in several cases, but the epithelial nature of the growth was proven by silver stains which brought out the typical alveolar arrangement of the cells. The question as to whether there is a sharp difference between lympho-epithelioma and transitional cell carcinoma seems to us to be of lesser importance because clinically there is very little difference in their development and essential characteristics. Both Ewing and Cappell have stated rather definitely that the points of similarity are greater than the points of differentiation. The weight of the evidence seems to point to the fact that both of these types originate in the transitional epithelium in the neighborhood of the lymphoid tissues of the throat and that the percentage of lymphocytic infiltration into the tumor mass depends on the abundance or scarcity of these lymphoid masses in the individual case. The essential features which characterize lympho-epithelioma are these:

1. The epithelial cells are definitely anaplastic, their protoplasm is pale, the cell boundaries vague with a tendency to syncytial formation. The nuclei are large, round or oval, clear and pale with several prominent nucleoli and frequent mitoses.

2. The cells tend to grow in sheets or columns, occasionally in anastomosing trabeculæ and frequently in such abundance as to suggest the appearance of Sarcoma. Silver stains, however, will bring out the interlacing supporting stroma and disclose the alveolar type of arrangement which in some cases is well marked.

3. Lymphocytic infiltration varies and in some instances may be so dense as to overshadow the epithelial elements. Careful examination of several sections will however soon disclose the typical pale epithelial cells described above.

The structure of transitional cell carcinoma as described by Cutler is shown to consist of masses of small round or polyhedral cells with large hyperchromatic nuclei occupying almost the entire cell. There is great variation in the size and staining qualities of the cells and they also tend to grow in anastomosing cords or sheets. Where the cells appear in great abundance they are apt to lose their epithelial characters and may closely resemble lympho-sarcoma.

In our series of cases it was this type of tumor which predominated (75%), whereas lympho-epithelioma was found much less frequently. The comparative infrequency of lympho-epithelioma in the nasopharynx was previously noted by Ewing, who in 300 tumors of the tonsil and nasopharynx found only 19 or 6.3% belonging to this group.

Reviewing what we have just said on the subject of nomenclature we feel that much could be gained through simplification of terminology, especially in view of the fact that the majority of these tumors run a characteristic course. The descriptions which we have read in the literature coincide to a remarkable degree with our own observations, which further confirm our opinion that transitional cell carcinoma of the nasopharynx presents a rather characteristic symptom complex. As early as 1922 we find that New[20] called attention to the early involvement of the cervical lymphatics and the symptomless progress of the primary disease which in so many cases was overlooked. Jovin as well as Ewing later described these neoplasms as "slowly growing tumors of moderate consistency growing expansively, at first slowly pushing aside the neighboring structures but later

infiltrating them." Cappell calls attention to the early appearance of large discrete glands often resembling lympho-sarcoma or Hodgkin's disease. The primary focus is often so small as to be completely overlooked. The fact that in the early stages the epithelial surface may not be broken led many of the older observers to deny the epithelial origin of the growth and was frequently pointed out as characteristic of endothelioma since they had no other way of accounting for the epithelial-like cells which predominated. Cutler laid particular stress on the early painless adenopathy, the characteristic firm consistency of the glands and their tendency to remain encapsulated and rather freely movable for a long time. Even long before these authors we find Heymann[21] quoting some of his predecessors as describing this slowly infiltrating growth as something entirely different from Sarcomas which were characterized by large tumor masses causing marked obstructive symptoms.

Symptoms

Having suggested that the development and growth of transitional cell carcinoma of the nasopharynx are responsible for a characteristic group of symptoms it behooves us to look into this phase of the subject more thoroughly. It is natural that an infiltrating tumor of the nasopharynx should produce a wide range of symptoms when one considers the numerous structures that lie in its path. A tumor that can grow about the eustachian tube, into the various basal foramina and through the orbital fissure, to say nothing of the nasal cavities and the pharynx proper, possesses protean possibilities to say the least. What interests us mostly is the question of early diagnosis and what symptoms may be looked for to suggest a diagnosis in suspected cases. It follows, therefore, that a knowledge of the principal locations where the tumor has its inception may prove of great assistance in determining the early symptoms. Unfortunately, very few of the reports we have read state explicitly just where the tumor was first located. Oppikofer mentions 11 cases in which the origin was traced to the lateral wall in 7 and the vault in 4. In Zuppinger's series of 32 cases the vault and lateral wall were involved in 9, the lateral wall alone in 7 and the vault alone in 6. Even in our series we find that in 9 cases (37%) the tumor was located merely in the "nasopharynx" without mention being made of the particular area involved. Six of

these, however, were large growths where it was impossible to determine the exact region first involved. Of the remaining 14 cases (60%), however, there is a definite record of the lateral wall (either eustachian orifice or Rosenmueller's fossa) being the seat of the growth in all but two. This, we believe, is very significant because a study of the records reveals a preponderance of symptoms referable to the ear and to branches of the fifth nerve. In addition to our own series we have analyzed 97 case reports including those of Zuppinger, Woltman,[22] Hansel,[23] Christianson[24] and Gardham,[13] and find the incidence of these symptoms as follows: Pain in the ear, tinnitus, impaired hearing or aural discharge appeared in 50% of the cases and as a rule were among the earliest subjective complaints. Pain in the distribution of the fifth nerve, exclusive of ear pains, was also found to be present in 50% of the cases. The character of the pain and its intensity was extremely variable. It was located in the temple, the forehead, the eye, the cheek, teeth and vertex. It is interesting to note that in numerous instances the pain was attributed to other causes than nasopharyngeal neoplasms and frequently was the basis of futile operations on other structures.

Cervical adenopathy, previously mentioned as an early symptom, was found in 35% of the collected cases. In our own series of 24 cases the presence of enlarged cervical or submaxillary glands was noted in 20 (80%). What is more remarkable is the fact that the adenopathy was present at the first examination in all but two of these cases. This observation certainly goes a long way toward corroborating previous testimony as to the prevalence of early glandular metastases which develop frequently without any subjective manifestation of the primary lesion.

These three symptoms, cervical adenopathy, tubal catarrh causing deafness and tinnitus and pain in the distribution of the fifth nerve are in our experience the most frequent and most significant findings in cases of infiltrating malignant tumors beginning in the lateral wall of the nasopharynx. Occurring alone or in combination they should direct the observer's attention to the region of the tubal orifice in the nasopharynx where in the majority of instances the focus will be found. This does not necessarily imply that there are no other symptoms. On the contrary, a whole array of manifestations ranging through the entire series of cranial nerves may be encountered in various cases depending on the direction and degree of extension cranial-

ward as well as on the size of cervical glands and the degree of pressure at the jugular foramen for which they may be responsible. It would be superfluous for us to go into detail as this phase of the subject has been more than adequately covered by Woltman,[22] New,[25] Burger,[26] and Roger and Paillas.[27] As a matter of fact, the appearance of symptoms referable to any of the cranial nerves beyond the first and second divisions of the fifth is at once an indication of extension through the basal foramina, the orbital fissure or the jugular foramen and heralds a stage of the disease that marks it practically hopeless. In the five autopsies which we had the opportunity of studying there was either extensive involvement of the base of the skull with attendant cranial nerve palsies or else widespread metastases in numerous vital organs. While these findings are instructive and interesting they do not possess the same practical bearing on the subject as a consideration of the early symptoms previously set forth. We have only to add this much: namely, that there is a small group of proliferative tumors chiefly Sarcomata or late carcinoma in which the early symptoms are those produced by the mass or bulk of the growth. We refer to nasal obstruction, nasal speech, impaired mobility of the soft palate and hemorrhage. In our series we found hemorrhage either nasal or postnasal noted six times, nasal obstruction five times and symptoms referable to impaired mobility of the palate twice. These symptoms naturally are significant when they appear and lead readily to the discovery of the malignant growth. It should be borne in mind, however, that they are characteristic only of a small group of cases, namely, Sarcomata and the late stages of Carcinomata and, therefore, are not to be considered of equal importance with the triad mentioned previously in connection with early diagnosis.

Summarizing briefly the symptomatology we may say that anaplastic or transitional cell carcinoma of the nasopharynx is responsible for three characteristic symptoms. These are unilateral painless cervical adenopathy, pain in the ear, deafness or tinnitus and pains referable to branches of the fifth nerve. It must be borne in mind that ulceration and hemorrhage are not early symptoms and must not be depended on for the diagnosis. A tumefaction that brings the suspected side into greater prominence than the opposite side should be sufficient to warrant a biopsy because these growths in the beginning are located beneath

the epithelial surface which in many instances is not invaded or broken through until quite late.

Symptoms due to choanal obstruction, such as impaired nasal breathing, nasal speech and impaired palate mobility, are due to tumors of large bulk, such as the advanced stages of carcinoma or the comparatively infrequent sarcoma.

Symptoms due to involvement of other cranial nerves aside from the fifth are evidence of infiltration through the basal foramina, orbital fissure or jugular fossa and indicate a late and hopeless stage of the disease.

Treatment

The malignant tumors of the nasopharynx are not amenable to surgical removal; consequently, the problem of treatment is entirely radiotherapeutic. The methods used in the treatment of these tumors are external radiation by means of X-rays or radium packs, combined in some instances with surface application of radium to the local lesion. Whether X-rays or radium packs are employed the underlying principle of treatment follows the technique established by Coutard. The primary lesion and the regional lymphatic areas are exposed daily or twice daily to X-rays or radium over periods varying between twenty days and sixty days. Adequate filtration is utilized in order to obtain the most highly penetrating rays. The total dose administered is estimated on a biological basis, the aim of which is to cause a complete regression of the growth without permanent damage to the surrounding normal structures. When the primary lesion is small and localized, surface applications of radium are combined with external radiation. The choice between high voltage X-rays and radium packs cannot be stated with certainty at the present time. The five year results of the former method are now well established. Whether or not the use of the penetrating gamma rays of radium will improve these results remains for the future to determine.

With rare exceptions the total dose of radiation does not vary with the estimated radiosensitivity of the tumor. As a rule the maximum dose compatible with the integrity of the tumor bed is administered.

Conclusions

1. In a series of 24 malignant tumors of the epipharynx, transitional cell carcinoma was found to be the most common type (75%) in the opinion of three pathologists.

2. In three cases the diagnosis of Sarcoma was considered, and in only one of these was there complete agreement. Our advisers differed on the other two.

3. Lympho-epithelioma was the diagnosis in six cases. Its similarity to transitional cell carcinoma in several cases was noted by our pathologists and the difficulty of differentiating them attributed to inadequate fixing and staining.

4. Endothelioma was mentioned but once by one and contested by two of the pathologists.

5. The early and characteristic symptoms of transitional cell carcinoma were found to be painless cervical adenopathy, tinnitus or deafness and pains due to involvement of the first and second branches of the trifacial nerve.

6. The majority of our cases originated in the region of the eustachian tube or lateral wall of the nasopharynx, thus accounting for the symptoms enumerated.

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HISTOLOGIC DESCRIPTIONS BY DR. OTTO SAPHIR

FIG. 1 — (Salinger and Pearlman) — Case 3168.

Jaffé: Anaplastic carcinoma
Cutler: Transitional cell carcinoma
Saphir: Epidermoid carcinoma

Sections reveal large epithelial cells with vesicular nuclei and eosinophilic cytoplasm. Marked anaplasia. Atypical mitotic figures. Occasionally, cells are more oval-shaped. Tendency toward keratinization, but no typical pearls.

The picture shows vesicular nuclei arranged in alveoli-like fashion.

FIG. 2 — (Salinger and Pearlman) — Case 5742.

Jaffé: Transitional cell carcinoma
Cutler: Transitional cell carcinoma
Saphir: Epidermoid carcinoma

Description similar to Case 3168 (Fig. 1). Picture reveals more oval-shaped cells. Anaplasia is marked.

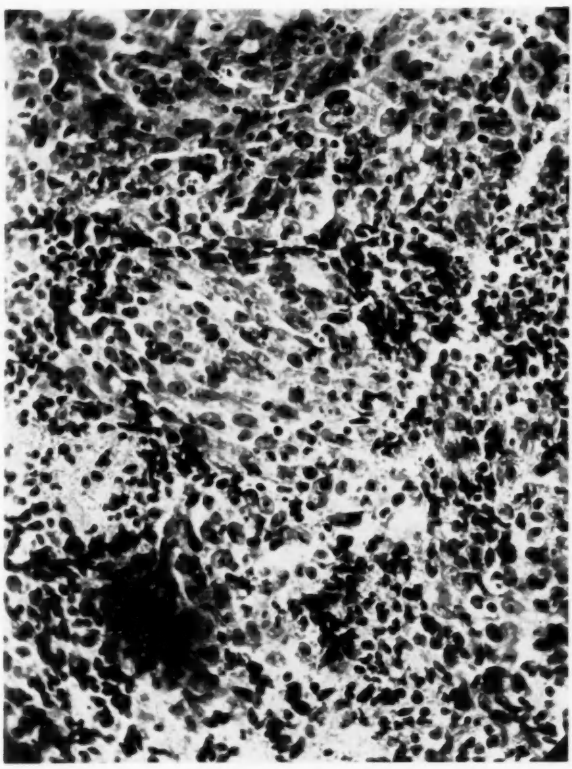

FIG. 3 — (Salinger and Pearlman) — Case 1667.

Jaffé: Transitional cell carcinoma
Cutler: Lympho-epithelioma
Saphir: Reticulum cell hyperplasia

The architecture of the lymph node is preserved. There are large foci consisting of reticulum cells showing evidence of marked hyperplasia. The germinal centers of the lymph node are also markedly hyperplastic.

NOTE: The reticulum cell stain would be essential to show the evidence of reticulum fibers which would definitely establish such a diagnosis. A possible epithelial nature of the cells can not be clearly demonstrated.

Picture reveals lymphoid structures. The center and the upper quadrants show reticulum cells which are more numerous than normal.

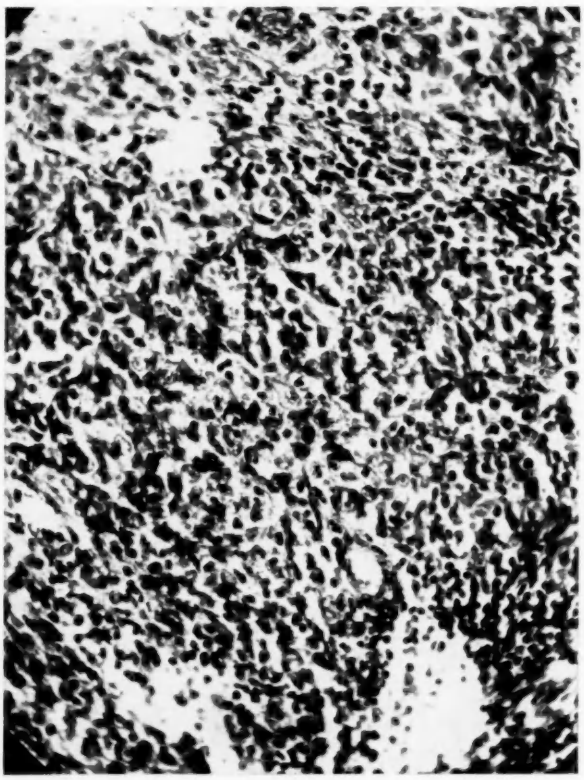

FIG. 4 — (Salinger and Pearlman) — Case 2937.

Jaffé: Squamous cell carcinoma
Cutler: Transitional cell carcinoma
Saphir: Transitional cell carcinoma

The tumor consists of one type of cell. The cell is oval-shaped with an elongated nucleus. Mitotic figures are numerous. There is no tendency toward keratinization.

Picture shows oval-shaped nuclei with mitotic figures. The absence of keratinization, the presence of one type of oval or slightly spindle-shaped tumor cells justify the diagnosis of transitional cell carcinoma.

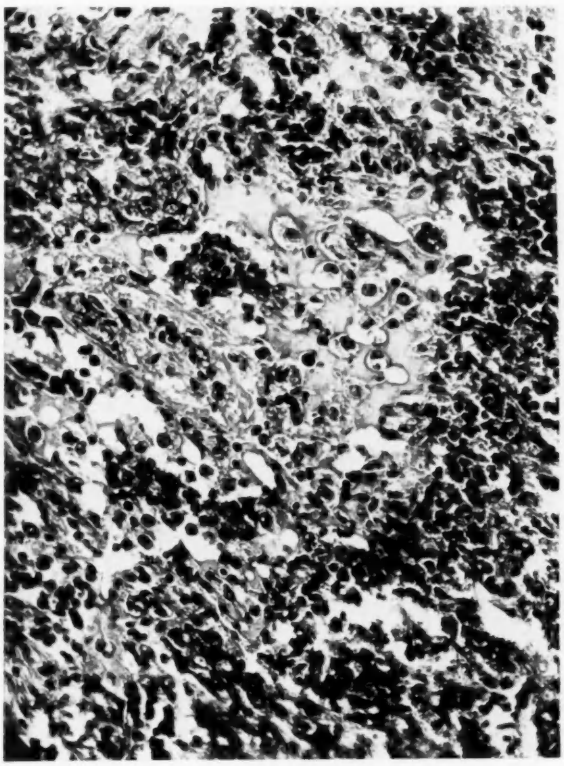

FIG. 5 — (Salinger and Pearlman) — Case 1256.

Jaffé: Transitional cell carcinoma
Cutler: Lympho-epithelioma
Saphir: Lympho-epithelioma

The tumor consists of sheets of large pale cells, most of which are round or oval. The cytoplasm is very indistinctly outlined. The nuclei are large, revealing distinct nucleoli. Between the cells a large number of lymphoid cells are present which seem to form a distinct part of the tumor. The absence of transitional or oval-shaped cells, the presence of the type of cells described and the intimate relation to the lymphoid cells are more in favor of lymphoid epithelioma than of that of transitional cell carcinoma.

Picture reveals the large tumor cells and shows the intimate relation between the epithelial cells and the round cells.

FIG. 6 — (Salinger and Pearlman) — Case 618.

Jaffé: Transitional cell carcinoma
Cutler: Transitional cell carcinoma
Saphir: Lympho-epithelioma

This is a badly stained slide. The type of cell present may be transitional in nature but the intimate relation between these cells and the lymphoid cells, the prevalent round nuclei seem more in favor of a lympho-epithelioma.

The picture reveals round- and oval-shaped epithelial cells and shows many lymphocytes between which epithelial cells are easily recognizable.

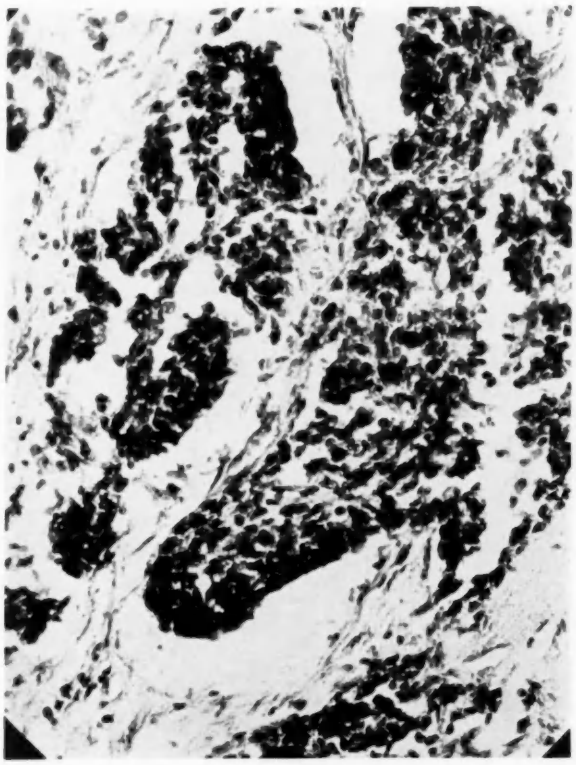

FIG. 7 — (Salinger and Pearlman) — Case 913.

Jaffé: Undifferentiated squamous cell carcinoma
Cutler: Transitional cell carcinoma
Saphir: Transitional cell carcinoma

Sections reveal much fibrous connective tissue and islets of epithelial cells. Many of these islets are fused. The epithelial cells show distinctly vesicular nuclei, many of which are of oval or transitional cell type. In a few fields the tendency towards formation of ill-defined cystic structure is recognized. Keratinization is absent.

NOTE: The type of cell and abortive formation of cysts (epithelioma adenoides cysticum) are more in favor of transitional cell carcinoma.

Picture shows groups of tumor cells. Note the oval and transitional cell type of cell.

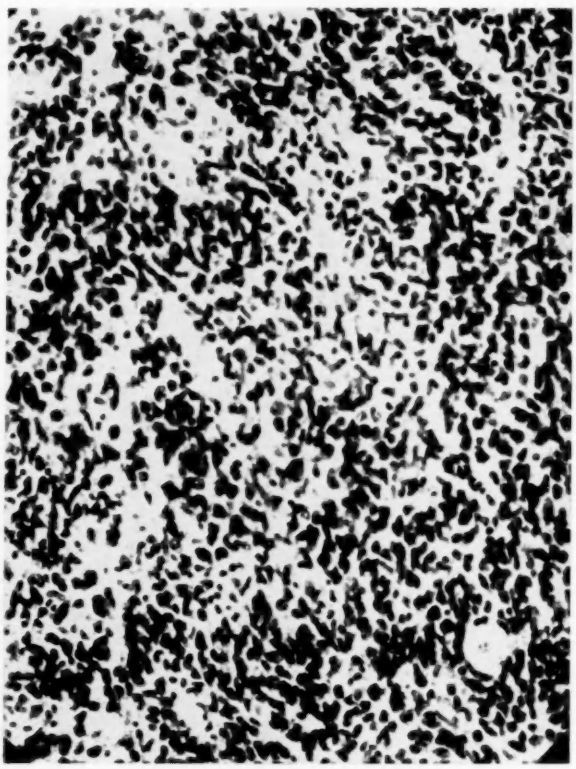

FIG. 8 — (Salinger and Pearlman) — Case 758.

Jaffé: Anaplastic carcinoma
Cutler: Transitional cell carcinoma
Saphir: Transitional cell carcinoma

The tumor cells show dark-stained nuclei with very little cytoplasm surrounding them. The shapes of the cells are oval and elongated. No tendency towards keratinization.

NOTE: While the anaplastic carcinoma can not be ruled out, the predominating type of tumor cell is more in favor of transitional cell carcinoma.

Picture is not at all characteristic and probably not taken from the representative field.

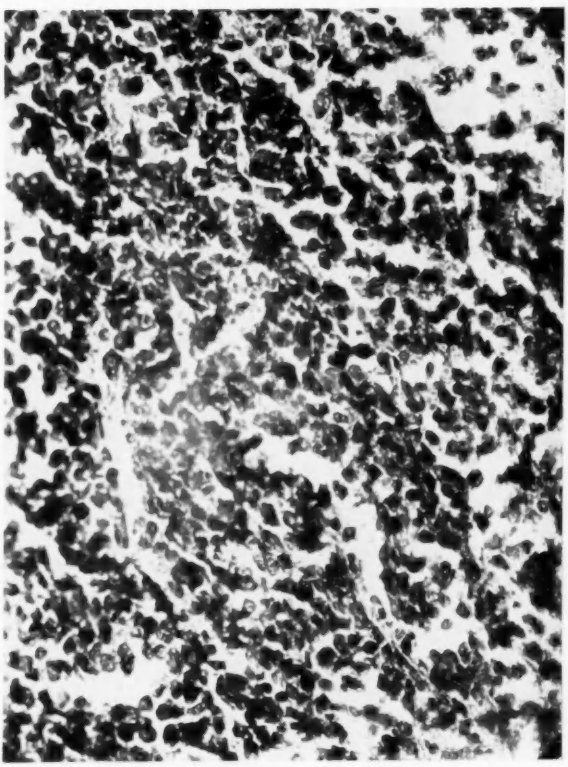

FIG. 9 — (Salinger and Pearlman) — Case 687.

Jaffé: Reticulum cell or lympho-sarcoma
Cutler: Transitional cell carcinoma
Saphir: Sarcoma (variety can not be ascertained)

The tumor consists of small round cells with hardly any recognizable cytoplasm. The nuclear membranes are ill-defined. Mitotic figures are numerous.

NOTE: This is apparently a sarcoma. The reticulum cell stain is essential to differentiate between lympho-sarcoma and the reticulum cell sarcoma.

Picture is very poor. The center is out of focus, yet the sarcoma cells are recognizable in the lower half of the picture, particularly the indistinct outline of the nuclear membranes.

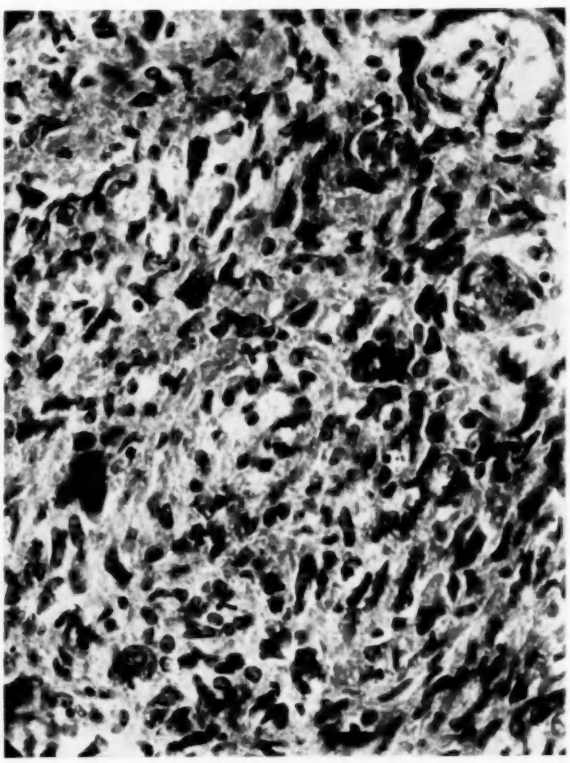

FIG. 10 — (Salinger and Pearlman) — Case 368.

Jaffé: Non-hornifying squamous cell carcinoma
Cutler: Transitional cell carcinoma
Saphir: Lympho-epithelioma? Hodgkin's disease?

Sections show many areas of necrosis and hyalinization, nucleoli accumulations of oval-shaped cells with vesicular nuclei and well outlined nucleoli. Many of the cells show two or three nuclei which are distinctly overlapping (characteristic of Dorothy Reed cells — Hodgkin's disease). Between the cells a few lymphocytes are seen.

Picture shows a multi-nucleated cell, a few large cells with prominent nucleoli and a number of lymphocytes.

NOTE: The various types present seem to speak for Hodgkin's disease or so-called Hodgkin's sarcoma. The fact that several types of cells are present is against transitional cell carcinoma. There is no indication that the tumor cells are squamous in nature, particularly since there is tendency towards keratinization. Lympho-epithelioma should be considered but it is more likely that this is a Hodgkin's disease or so-called Hodgkin's sarcoma.

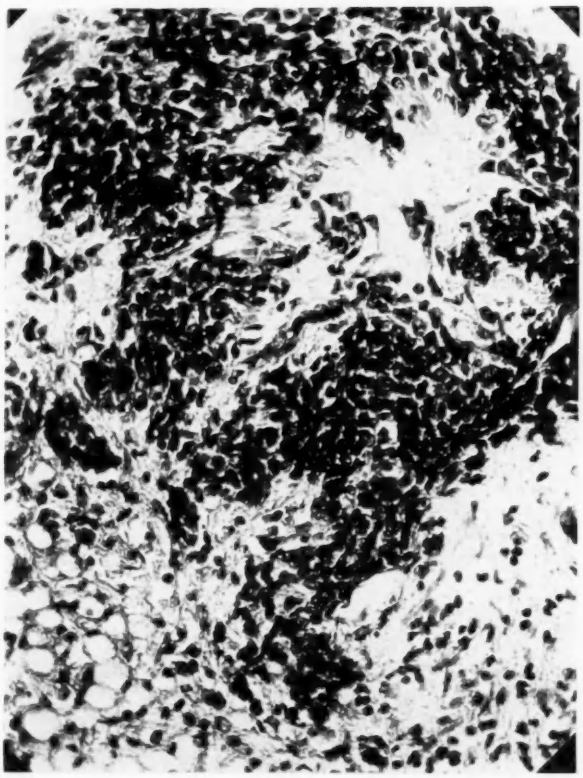

FIG. 11 — (Salinger and Pearlman) — Case 1104.

Jaffé: Transitional cell carcinoma
Cutler: Transitional cell carcinoma
Saphir: Transitional cell carcinoma

(Metastases to the liver).

Picture which is taken from the slide that is markedly overstained shows the transitional cells.

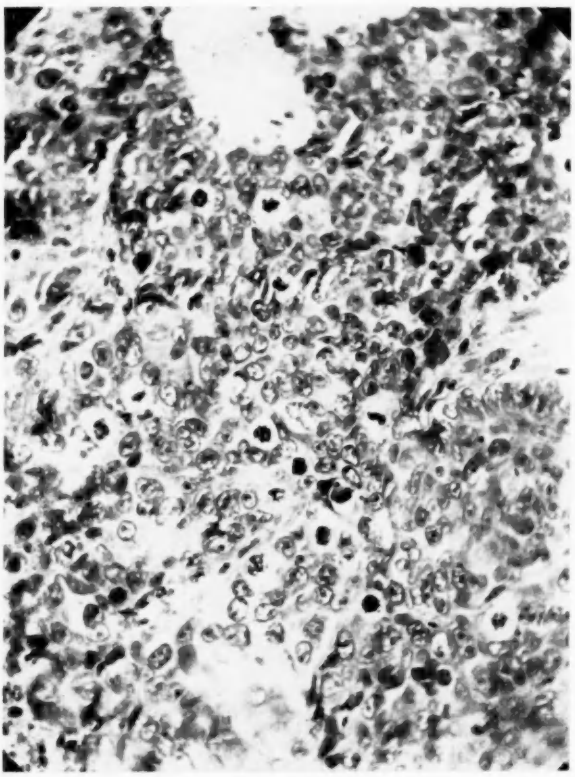

FIG. 12 — (Salinger and Pearlman) — Case 5523.

Jaffé: Anaplastic carcinoma
Cutler: Transitional cell carcinoma
Saphir: Squamous cell carcinoma

The cells are large, showing vesicular nuclei and oval cytoplasm. There is a tremendous number of atypical mitotic figures. There is a tendency towards keratinization. There are no definitely formed pearls recognizable. Mitotic figures are extremely atypical.

NOTE: The type of cell, the type of mitotic figure and quality of the latter are more in favor of squamous cell carcinoma even though no atypical horn pearls are recognizable.

Picture shows beautifully the type of tumor cells and the large number of atypical mitotic figures.

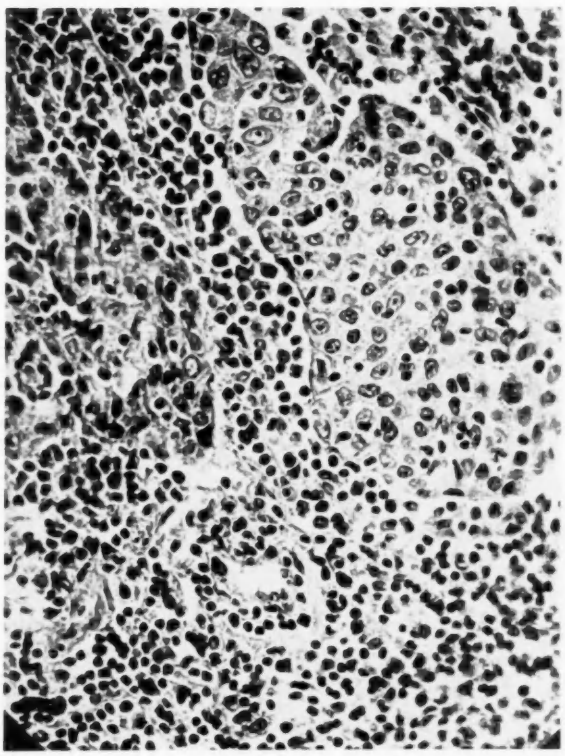

FIG. 13 — (Salinger and Pearlman) — Case 6289.

Jaffé: Transitional cell carcinoma or lympho-epithelioma
Cutler: Transitional cell carcinoma or lympho-epithelioma
Saphir: Transitional cell carcinoma or lympho-epithelioma

The type of epithelial cells present and their intimate relation to lymphoid cells are in favor of lympho-epithelioma.

Picture shows both the "lympho" and "epithelioma" parts very clearly.

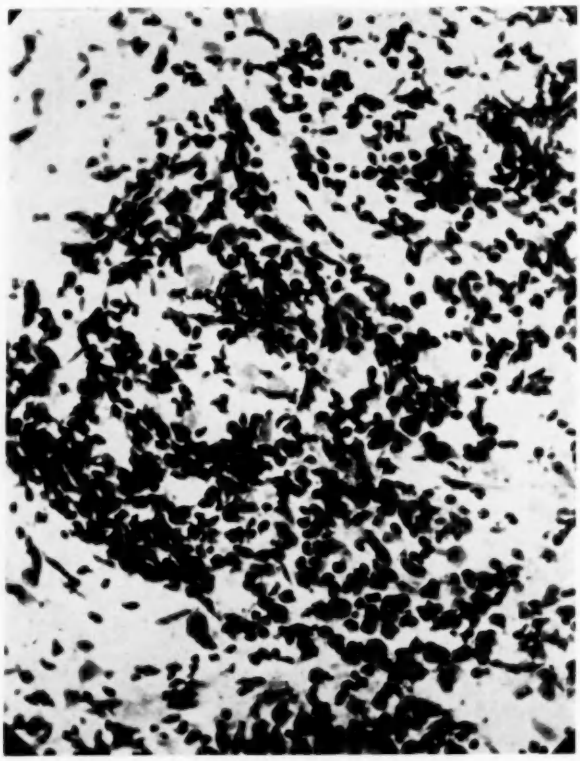

FIG. 14 — (Salinger and Pearlman) — Case 23.

Jaffé: Squamous cell carcinoma
Cutler: Transitional cell carcinoma
Saphir: Transitional cell carcinoma

Picture shows typical transitional cells which are markedly overstained.

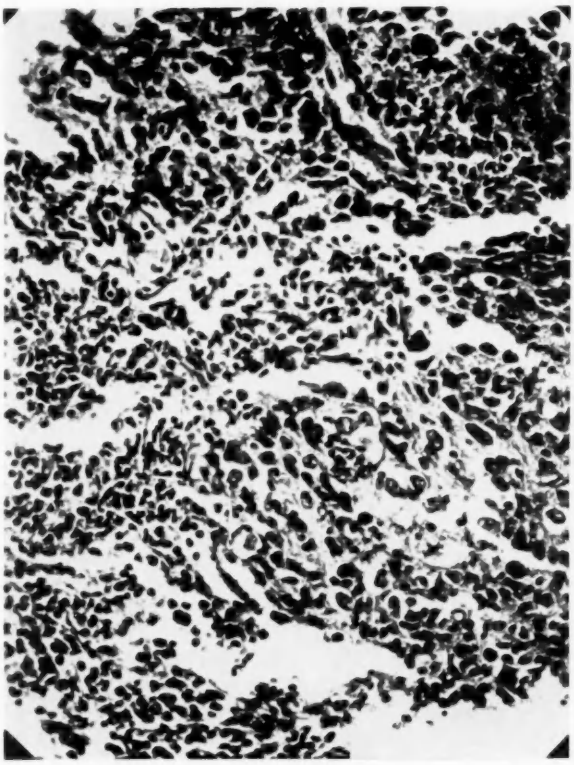

FIG. 15 — (Salinger and Pearlman) — Case 2868.

Jaffé: Transitional cell carcinoma
Cutler: Transitional cell carcinoma
Saphir: Transitional cell carcinoma

The type of tumor cell is quite characteristic of transitional cell carcinoma.

Picture is not taken from a characteristic field. In the upper portion a few dark-stained transitional cells are seen.

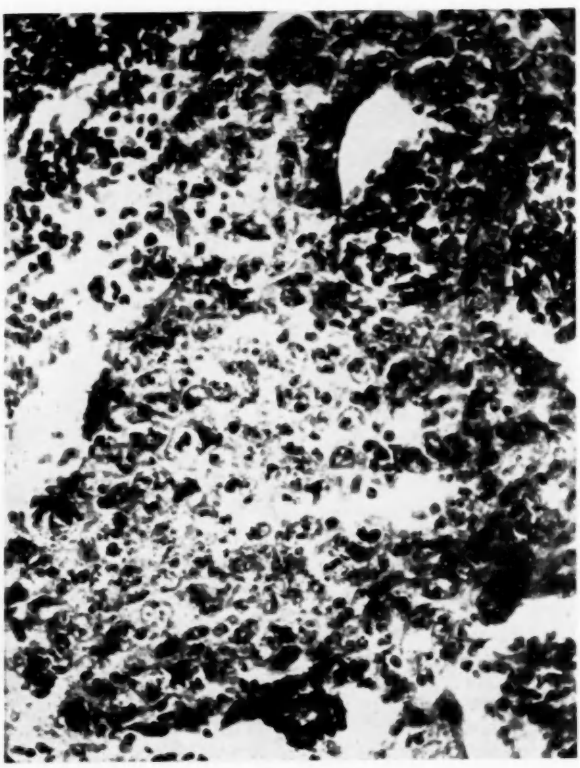

FIG. 16 — (Salinger and Pearlman) — Case 1327.

Jaffé: Transitional cell carcinoma
Cutler: Transitional cell carcinoma
Saphir: Epidermoid (transitional?) cell carcinoma

In several fields it is difficult to decide whether this is a transitional cell or epidermoid carcinoma. The latter seems indicated by the presence of tumor cells with vesicular nuclei which are found, while the former is indicated by the presence of many spindle-shaped or oval-shaped cells.

Picture is not at all characteristic.

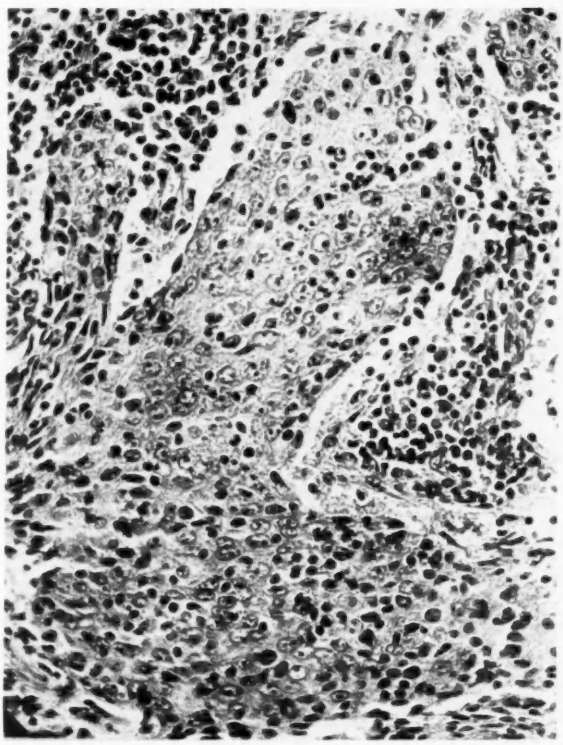

FIG. 17 — (Salinger and Pearlman) — Case 1309.

Jaffé: Transitional cell carcinoma
Cutler: Transitional cell carcinoma
Saphir: Transitional cell carcinoma

It is difficult to decide whether this is a lympho-epithelioma or transitional cell carcinoma. The intimate relation between the epithelial cells and the lymphocytes is more in favor of lympho-epithelioma. Transitional cell carcinoma can not be ruled out.

The picture seems more characteristic of a lympho-epithelioma.

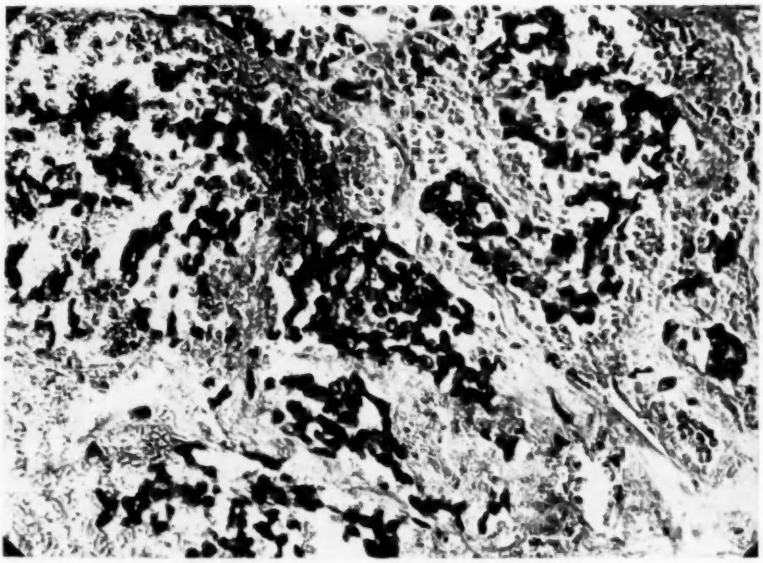

FIG. 18A — (Salinger and Pearlman) — I. F.

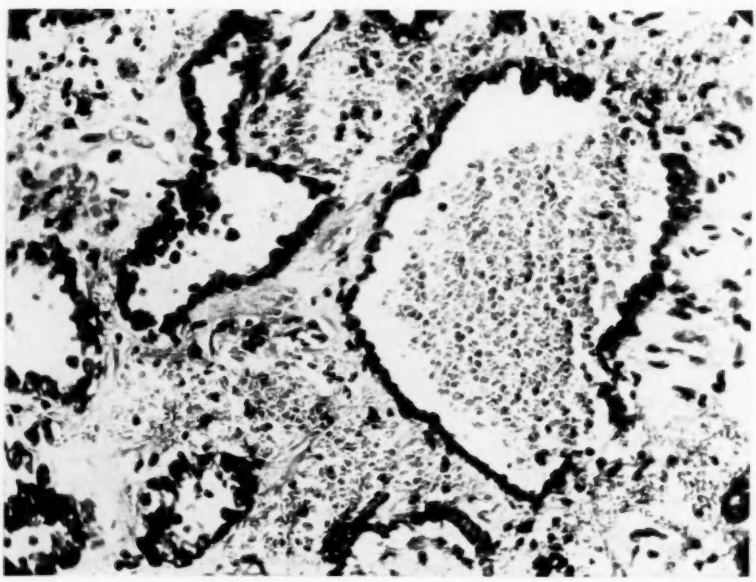

FIG. 18B — (Salinger and Pearlman) — I. F.

Jaffé: Basal cell carcinoma invading the blood vessels.
Cutler: Adenoid cystic epithelioma
Saphir: Endothelioma

These cells are arranged in clusters and channels surrounding small blood spaces. In places they proliferate, obliterating the lumen. The blood spaces are either empty or contain a few red corpuscles. Because the spaces described are obviously vessel spaces and not the result of cystic degeneration this is an endothelioma rather than an epithelioma adenoides cysticum.

Pictures show (Fig. 18A) clusters of cells; (Fig. 18B) vessel spaces.

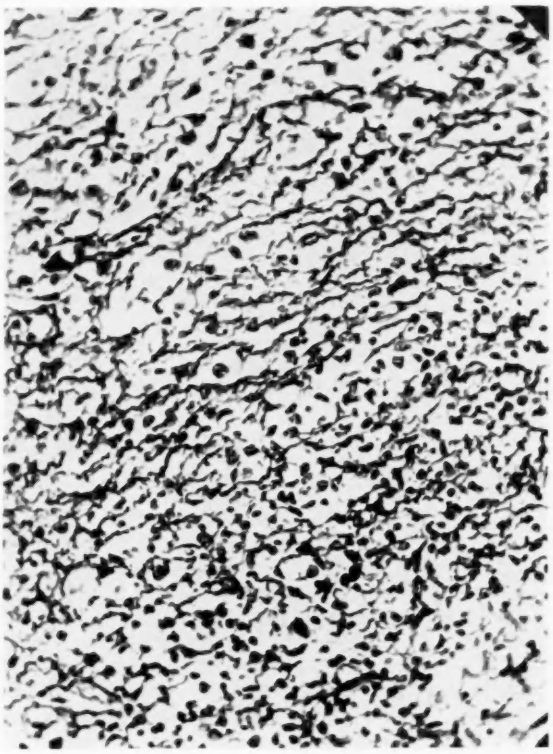

FIG. 19 — (Salinger and Pearlman) — Case 32P.

Jaffé: Reticulum cell sarcoma
Cutler: Spindle and polyhedral cell sarcoma
Saphir: Spindle and polyhedral cell sarcoma

The bulk of the section consists of spindle and polyhedral cells which show variation in size, shape and staining quality. The reticulum stain reveals a moderate amount of reticulum which is indicative of the sarcomatous nature of the tumor.

Picture reveals the reticulum.

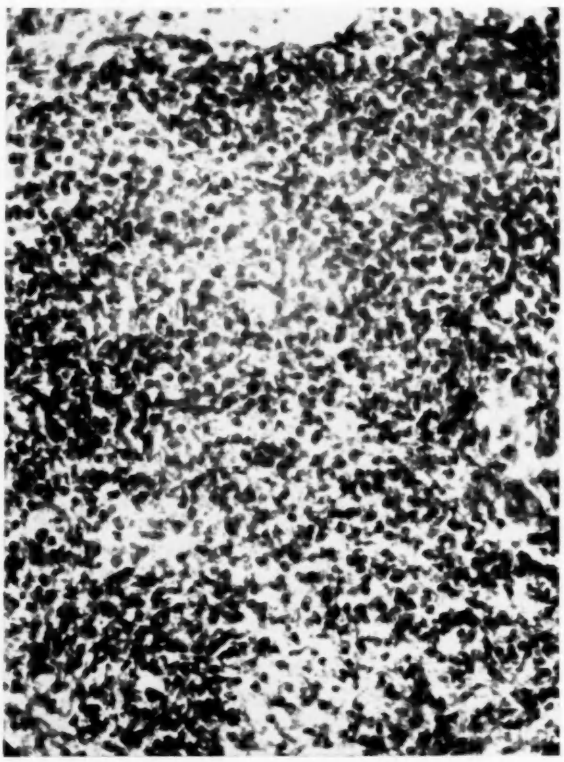

FIG. 20 — (Salinger and Pearlman) — H. O.

Mayo: Epithelioma Grade IV

Jaffé: Transitional cell carcinoma?

Cutler: Lympho-epithelioma or transitional cell carcinoma

Saphir: Lympho-epithelioma or transitional cell carcinoma

This is a badly-stained slide. Most of the cells are spindle or oval-shaped, showing a number of mitotic figures. A moderate amount of lymphoid cells is also present. It is more likely that this is a transitional cell carcinoma than the lympho-epithelioma, although the latter can not be ruled out. The oval type of cell, however, dominates the histologic picture. The figure is very indistinct.

Histologic Diagnoses

Identification	Sex-Age	*Jaffé*	*Saphir*	*Cutler*
1340	M. 44	Transitional Cell Carcinoma	Lympho-epithelioma	Transitional Cell Carcinoma
3168	F. 21	Anaplastic Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma
5742	M. 41	Transitional Cell Carcinoma	Epidermoid Carcinoma	Transitional Cell Carcinoma
5864	M. 65	Non-hornifying Squamous Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma
1667	M. 10	Transitional Cell Carcinoma	Reticulum Cell Hyperplasia	Lympho-epithelioma
2937	M. 58	Squamous Cell Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma
618	M. 60	Transitional Cell Carcinoma	Lympho-epithelioma	Transitional Cell Carcinoma
913	M. 70	Undifferentiated Squamous Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma
758	M. 33	Anaplastic Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma
687	M. 42	Reticulum Cell Lympho-Sarcoma	Sarcoma	Transitional Cell Carcinoma
368	F. 35	Non-hornifying Squamous Carcinoma	Lympho-epithelioma	Transitional Cell Carcinoma
1104	M. 41	Transitional Cell Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma
5523	M. 60	Anaplastic Carcinoma	Squamous Cell Carcinoma	Transitional Cell Carcinoma
6289	M. 51	Transitional Cell Carcinoma or Lympho-epithelioma	Transitional Cell Carcinoma	Transitional Cell Carcinoma or Lympho-epithelioma
23	M. 31	Squamous Cell Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma
2868	F. 58	Transitional Cell Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma
1327	M. 19	Transitional Cell Carcinoma	Epidermoid Transitional Cell Carcinoma	Transitional Cell Carcinoma
1309	M. 32	Transitional Cell Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma
667	M. 51	Epidermoid Carcinoma	Reticulum Cell Sarcoma	Epidermoid Carcinoma or Sarcoma?
I. F.	F. 42	Basal Cell Carcinoma invading Bl'd-Vessel	Endothelioma	Adenoid-Cystic Epithelioma
32 P	M. 17	Reticulum Cell Sarcoma	Spindle and Polyhedral Cell Sarcoma	Spindle and Polyhedral Cell Sarcoma
H. O.	M. 45	Transitional Cell Carcinoma	Transitional Cell Carcinoma or Lympho-epithelioma	Transitional Cell Carcinoma or Lympho-epithelioma
1517	M. 42	Transitional Cell Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma
R. U. S.	M. 20	Transitional Cell Carcinoma	Transitional Cell Carcinoma	Transitional Cell Carcinoma

Summary of Histologic Diagnoses	*Jaffé*	*Saphir*	*Cutler*
Anaplastic or Transitional Cell Carcinoma	19	14	18
Transitional Cell Carcinoma or Lympho-epithelioma	1	1	2
Squamous Cell Carcinoma	2	1	0
Sarcoma	2	3	1
Lympho-epithelioma	0	3	1
Other Diagnoses	0	2	2
Total	24	24	24

Summary of Clinical Histories

Slide No. Sex-Age	Duration Symptoms Onset to 1st Exam.	Location of Tumor	Glands	Ear Symptoms	Fifth Nerve Symptoms	Other Cranial Nerves	Treatment	Result
1327—M. 19	8 mo.	Right eustachian orifice		Pressure. Impaired hearing.	Headaches. Frontal and occipital pains.		Ra. Bomb 100,000 mg. hrs. Also local plaque.	Growth disappeared. No evidence of tumor 1 yr. later.
2868—F. 58	Several Mos.	Vault and right side	Movable, tender at angle of jaw.	Tinnitus. Impaired hearing.			Ra. external; also local plaque.	Still under treatment.
1517—M. 42	10 mo.	Right Rosenmueller. Fungating mass	Angle of jaw. First symptom.	Tubal Catarrh. Pain.	Headache.	Paralysis, soft palate.	Ra. bomb.	Still under treatment.
Rus—M. 20	6 mo.	Nasopharynx	6 months. Early suppuration.	Earache 3 months.	Pain in head.		Ra. bomb. 135,000 mg. hrs.	Temporary regression. Died.
32 P—M. 17		Large mass filling nasopharynx	Bilateral large.		Neck and head pains.		Ra. bomb 32,000 mg hrs. Local tandem tubes.	General metastases. Died.
1667—M. 10	6 wks.	Vault	Large smooth. Earliest symptom. Dissected out.	Discharge.			Ra. collar 5,400 mg. hrs. Plus needles.	Alive and well 1 yr. later.
5742—M. 41	12 mo.	Right eustachian tube	1 yr. Earliest symptom. Bilateral.	Tinnitus 2 months.			Ra. collar 15,000 mg. hrs. Ra. seeds.	Died.
5864—M. 65	4 mo.	Nasopharynx	Large, firm mass.		Severe in head and neck.	Hoarseness. Difficulty swallowing. Exophthalmos.	Refused treatment.	Left hospital.
2937—M. 58	Over 1 yr.	Left side	Large, firm bilateral.	Pain. Tinnitus. Impaired hearing.	Pain in eye, neck, head & shoulder.	(Ext. rectus) V, VI, X and sympathetic.	Radium.	Under treatment. Poor condition.
3168—F. 21	14 mo.	Large mass nasopharynx	Large, firm. Ant. & Post. triangles. 7 mo.			Exophthalmos; ex. rectus and sup. oblique.	No record.	Unknown.
1340—M. 44	4 mo.	Large mass mostly to right side	Large unilateral.	Discharge, Pain.	Pain in jaw, head and face.	Ext. rectus.	9 needles each 1.3 mg. 4 days.	Died.
1309—M. 32	3 mo.	Right side fleshy irregular mass		Deafness 6 weeks.			Ra. bomb 160,000 mg. hrs.	No evidence of tumor 6 mos. later.

Summary of Clinical Histories

Slide No. Sex-Age	Duration Symptoms Onset to 1st Exam.	Location of Tumor	Glands	Ear Symptoms	Fifth Nerve Symptoms	Other Cranial Nerves	Treatment	Result
618—M. 60	2 mo.	Large mass filling nasopharynx	Bilateral cervical and submaxillary.		Pains in mouth, neck and head.		No treatment.	Left hospital.
5523—M. 60	3 mo.	Nasopharynx	Rt. submaxillary first symptom.	Discharge later.			Deep X-rays.	Died.
6289—M. 51	8 mo.	Left fossa Rosenmueller	Cervical first symptom.				Deep X-rays.	Well 18 months later.
687—M. 42	2 mo.	Large mass filling nasopharynx, lat. walls, tonsils.	Bilateral. Rapid Development. Freely movable.	Pain.	Pains in head and neck.	Late involvement I, III, IV, V, VI.	Deep X-rays.	Temporary improvement. Relapse. Died.
368— F. 34	12 mo.	Large mass nasopharynx	Large bilateral. Early symptom.	Deafness.	Severe Head pains.	Ext. rectus.	8,400 mg. hrs.	Died.
913—M. 70	10 mo.	Right eustachian tube	Large.	Impaired hearing.	Head pains.	Ptosis. Ext. rectus. Facial paral.	X-rays 3 times w'kly for 7 months.	Died.
758—M. 33	Several Mos.	Posterior wall. Large fungating	Bilateral cervical inguinal axillary.		Head pains.		X-rays.	Died.
1104—M. 41	12 mo.	Soft ulcerating mass. Rather large	Bilateral painless. Incised; diag. followed.			Pain in spine. Paralysis lower extremities.	No treatment.	Died.
667—M. 51	3 mo.	Right side firm	Unilateral early.		Pains in head and face.		Ra. bomb 290,000 mg. hrs.	Temporary improvement. Died.
H. O.—M. 45	4 mo.	Left nasopharynx	Bilateral firm.	Stuffy.	Pains in eye, head and face.	Proptosis ext. rectus.	Ra. bomb 56,000 mg. hrs.	Temporary improvement.
23—M. 31	Months			Tinnitus early. Deafness.	Pains in eye, temple and neck		No treatment.	Left hospital.
I. F. — F. 42	* See below	Peanut size growth roof of nasopharynx, right side.		Pains. Impaired hearing.	Pains in head and neck.		Ra. bomb 60,000 mg. hrs.	Still under treatment.

* 1927, adenoid removed; diagnosed Ca. Diathermy. Primary cure. Recurrence.

Summary of Autopsy Reports

SLIDE NO.	SEX-AGE	DATE OF ADMIS'N	DATE OF DEATH	PRINCIPAL SYMPTOMS	DESCRIPTION OF TUMOR	METASTASES	HISTOLOGIC DIAGNOSES
687	M. 42	Jan. 22, 1933	June 27, 1933	Choanal obstruction. Mass extending down to tonsil region. Bilateral ptosis. Involvement of I, III, IV, V, VI	Fills nasopharynx. Roof destroyed. Sella and hypophysis invaded. Clivus, roof of sphenoid, left major and minor wings with adjacent squama and tip of petrous destroyed.	Nodule in left adrenal. Same structure as original tumor. Bilateral cervical glands.	*J.*—Retic. Cell Lymphosarc. *S.*—Sarcoma *C.*—Transitional Cell Ca.
368	F. 58	Feb. 17, 1934	April 1, 1934	Nasal hemorrhages. Deafness. Diplopia. Hemicrania.	Vault and lateral walls involved Area measures 30x35 mm.	Large cervical glands. No other metastases. Suppurative sphenoiditis and meningitis cause of death.	*J.*—Non hornifying Sq. Ca. *S.*—Lympho-epithelioma *C.*—Transitional Cell Ca.
913	M. 70	Aug. 18, 1933	Sept. 19, 1933	Deafness, hemicrania, ptosis, facial paralysis.	Surrounds eustachian tube destroying its walls. Invasion foramen lacerum to base of brain. Surrounds knee of int. carotid. Extends laterally under dura along ant. aspect temporal bone.	Nodes in liver, cervical glands lung, frontal lobe of brain, third lumbar vertebra.	*J.*—Undifferentiated Sq. Ca. *S.*—Transitional Cell Ca. *C.*—Transitional Cell Ca.
758	M. 33	June 14, 1933	July 24, 1933	Bilateral cervical glands, inguinal and axillary. Head pains. Backache.	Ulcerating mass vault posterior and lateral walls.	Cervical, supraclavicular, peri-aortic, peri-iliac, peri-pancreatic, peri-gastric and inguinal glands. Liver, spleen, adrenals, peritoneum and pelvis.	*J.*—Anaplastic Carcinoma *S.*—Transitional Cell Ca. *C.*—Transitional Cell Ca.
1104	M. 41	Sept. 26, 1934	Nov. 2, 1934	Bilateral cervical glands, suppuration and incision. Pain, lower spine. Paralysis of legs, loss of sensation from toes to neck.	Large soft mass filling nasopharynx.	Cervical glands, ribs, dorsal and lumbar spine, left pubic bone.	*J.*—Transitional Cell Ca. *S.*—Transitional Cell Ca. *C.*—Transitional Cell Ca.

DISCUSSION

Dr. Gordon B. New, Rochester, Minn.: In 1920, I reported forty-six patients with malignant tumors of the nasopharynx and described the syndrome that they presented, emphasizing the fact that it was necessary to make a very careful examination of the nasopharynx in order to find the small lesions and that the finding of an epithelioma in the nasopharynx frequently cleared up many indeterminate diagnoses. In 1931, at the Academy meeting at French Lick, I reported 246 of these cases, emphasizing at that time the neurologic findings associated with them.

There has been a great deal of discussion as to the terminology of these tumors among pathologists but it is really of little clinical value. As to the classification of these highly malignant growths, I can only say that Dr. Broders, after having studied 267 squamous cell epitheliomas and 78 lymphosarcomas of this particular region, came to the conclusion that these tumors were squamous cell epithelioma, grade four. He felt that their identity with squamous cell tumors should be maintained and that it was superfluous to try to create an additional name for growths when there is no sound basis for doing it.

Regarding the diagnosis of endotheliomas and branchiogenic carcinoma, occasionally made in this type of lesion, we quote Saareste, who regarded endothelioma as a "refuge of the destitute" which has too often been resorted to when a malignant neoplasm of the upper air passages could not be definitely classified as carcinoma, sarcoma, or carcinoma (mixed tumor type). Hudson pointed out that writers in the past have erroneously attributed a branchiogenic origin to squamous cell epithelioma found in the neck near the angle of the jaw. Most of these undoubtedly were metastatic from lesions primary to obscure situations in the upper respiratory tract.

In the treatment of these highly malignant neoplasms of the nasopharynx, we have applied radium directly into or against the tumor and used x-ray therapy externally over the head and neck. Of all the traced treated cases of lymphosarcoma, the average life in months was 34.5. The average life in months of the cases that are still alive was 75.5. Of the squamous cell epithelioma, grade four, the average life in months of all the traced treated cases was 22.2 and the average life in months of the living cases was 19.7. The average life in months of the cases that were not treated was 5.2 for lymphosarcoma and 7.2 for the squamous cell epithelioma, grade four. Of the traced cases of all the treated highly malignant tumors in this region, 46.8% of the lymphosarcomas lived over three years and 34.3% of them lived over five years. Of the squamous cell epithelioma, grade four, 17.1% lived over 3 years and 9.3% lived over five years. It is readily seen from these figures that radiation accomplishes a great deal in prolonging the life in patients with highly malignant tumors of the nasopharynx.

DR. SAMUEL SALINGER, closing: I am very grateful to Dr. New for his discussion. Naturally, we have the highest regard for the work that Dr. New has done and ascribe the utmost value to his statistics, which we know have been most carefully compiled. His results agree fairly well with those of the other authors who have been employing radiotherapy.

The cases we have been treating in the last two years are beginning to show some improvement. The whole question that we attempted to solve here, or at least to clarify, was, first, the nomenclature, and second, to bring to a focus an appreciation of a definite type of tumor which we believe has been frequently overlooked. While it is true that this matter has been touched upon previously — Dr. New did it in 1921 or 1922 and it has since then been repeated — it is a subject that can bear repetition.

In the paper as it will be published, there will appear certain details of the case histories which will show how frequently the nasal pharyngeal lesion was overlooked and how the patient was treated for enlarged glands, presumably of tubercular nature, and how abscesses of the neck were opened without any intimation of the original lesion.

USE OF GELATIN PROSTHESES IN FACIAL RESTORATION

OSCAR V. BATSON, M.A., M.D.

PHILADELPHIA

WHEN THE features have been destroyed by disease, through accident or by therapy, to such a degree that the patient feels himself disfigured in the eyes of others, it is the duty of the physician to remove, if possible, the real or fancied stigma. Plastic surgical procedures should always be considered first in these restorations. However, when the vitality of the skin has been lowered through age, disease or radiant therapy, it is not always possible to use surgical measures. Moreover, in some conditions, such as in the loss of an ear, the art of the plastic surgeon is not yet advanced to the point where a wholly satisfactory restoration is possible. A prosthesis affords a means by which the patient may feel sufficient mental ease that he is willing fully to re-engage in his activities.

Many substances, such as ivory, vulcanite and metal, have been used in these efforts at restoration and various devices have been used to hold the finished prosthesis in place. The principal objection to all rigid prostheses is that they do not move with the surrounding features and that they have a visible edge. As a result, it is only with the greatest stretch of imagination that the prosthesis made of rigid material can be thought to have a natural appearance. It is merely a mask for the deformity.

Gelatin, when properly prepared with glycerin and suitably colored, makes an ideal prosthesis. Since it can be made to adhere to the skin with a cement, no mechanical support is necessary. The edges can be made so thin that the prosthesis blends imperceptibly with the features. The material is flexible and moves with the changing expression. In many patients the gelatin prosthesis is not obvious to the average observer at conversational distance.

In its very nature the gelatin prosthesis is a temporary affair. The patient is supplied with proper molds, a suitably colored gelatin mass, necessary cement, and advised as to the best methods for rendering the prosthesis inconspicuous. The entire outfit is so small as to be carried about easily if necessary, and the technical ability required is practically nil.

The entire matter can probably best be understood by describing the serial steps followed in handling a case. Obviously, the first consideration is whether in the given case plastic surgery is unsuited. The next is whether or not the patient is sufficiently interested in his personal appearance to make the use of the prosthesis satisfactory. For example, I do not feel that a patient who is careless about personal appearance would make a suitable case for a gelatin prosthesis. Such a patient would probably be perfectly content to wear a bandage over the disfigured part or to wear a prosthesis made of some rigid material which merely acts as a screen for the deformity.

In preparing for actual work the following are desirable: a cast of the patient's features including the general symmetry of the head, which may be studied (for the restoration of the ear this is not so essential), an accurate cast of the area which the prosthesis must fit; full face and profile photographs as well as a photograph of the patient before disfigurement, if that is possible. As to the casts, the large cast can most conveniently be made in one of the commercially obtainable agar agar containing materials, or it may be made in the mass prepared according to the following formula:

Agar	25	grams
Water	750	cc.
Cellucotton	2.5	grams
Zinc oxide (artists')	25	grams

This material or the material obtained commercially is melted and allowed to cool to slightly above body temperature. For the first coat, it is applied to the features with a brush and additional coats are added with an ear syringe or with a spoon. The mold is made about an inch thick and allowed to harden. This is readily removed from the face. A positive is then prepared by painting on melted white beeswax, which may be suitably colored. In my opinion, no material for moulages equals beeswax suitably colored with its surfaces slightly powdered. Masses can also be made of beeswax, resin and powdered silex and various backing materials, such as gauze, may be used.

In addition to the mask as made above, I also take a small impression in plaster, using brushless shaving cream as a separating medium; I also make a positive in dentists' model stone. I do this because I have had experience with slight warping of casts made by the other method. On this small stone model the technical features of the prothesis are worked out. Here there are many things to be considered and no specific directions can be given. A nose affords certain technical problems and some of these will be pointed out. It is necessary to provide the patient with adequate air-ways and it is further necessary to make the artificial organ so as to avoid snores or whistles. Ease of casting a prosthesis must also be considered at this time, for a mold must be produced that will not be troublesome to the patient. For the ear, a three-piece mold has been commonly used. However, I am here demonstrating a satisfactory two-piece mold for the ear. I feel that the patient should be given a mold of not more than two pieces. When these air-ways and attachment points have been decided upon, the small model is accordingly altered and by the usual methods a new model is made which contains these points. This model is made very carefully with the idea that it will be one part of the final mold. The missing part is now modelled freehand in such a way that the maximum amount of actual skin remains exposed; the juncture lines of the prosthesis with the skin should be as inconspicuous as possible, also the prosthesis should have adequate structural rigidity. Great care must be taken to feather out the model to an imperceptible thinness so that the material will blend readily with the skin. Frequently, special problems arise at this stage. For example, in one of the cases shown, because of the displaced ala, it was necessary to widen the entire nose to a greater width than at first would seem advisable. However, this must be done in such a manner that the total result is acceptable to the eye. This is managed by constantly applying the part that is being modelled to the wax moulage so that the part may be blended with the general contour of the features. When the modelling is finished satisfactorily the surface is broken up into pits which simulate skin markings. These may be somewhat exaggerated on the original. From this model, affixed to the mold base, dental casting stone is poured, again using brushless shaving cream as a supporting medium. This, when set, completes the molds. The next step is the pouring of a trial prosthesis. The halves of the molds are separated, the modelling material removed, and a glycerin jelly melted.

I have found that there are but three things essential to the basic gelatin mass: gelatin, water and glycerin. The water is largely useful only for the initial preparation. I make up the fundamental gelatin mass as follows:

Best gelatin	25 grams
Best glycerin	25 to 37.5 grams
Water	100 cc.

A smaller quantity of glycerin gives a harder mass suitable for summer climate, while the larger amount of glycerin gives greater elasticity and is suitable only for cooler weather. The ingredients above are allowed to stand for a few minutes and then are melted in a double boiler. The mass is poured out into thin sheets and allowed to remain for several days in a dry place so that the water largely evaporates. Color is added which is suitable to the patient's complexion and the material is ready for use.

Some material prepared in this manner is poured into the molds as described above, after the inner surface of the molds have been covered with brushless shaving cream as a separating medium. When the mass has solidified, the molds are separated and the prosthesis removed. It will be found that the gelatin has feathered out into a very thin edge. This is torn free. The trial prosthesis is examined then for defects in structure which have not been anticipated. The defects can commonly be changed by carving the mold suitably. The prosthesis is then prepared in suitable color. The back of it is coated with theatrical spirit gum and then tried on the patient. Here again it is sometimes found that errors have been made. Perhaps, if the part is a nose, a whistle may be present, or perhaps the point of attachment is not secure. Sometimes these can be corrected on the mold, sometimes a new model has to be made. Occasionally the part is not satisfactory to the patient who has some ideas as to how he would like to look. This stage is most trying to both patient and doctor. The patient has to accommodate himself to an entirely new condition. After experience with a few patients one realizes this stage and learns to handle the situation. Frequently, after the changes suggested have been made, the patient will return to the original model.

When the model is satisfactory, duplicate molds are made. The patient is instructed in the making of the casts, is provided with suitably colored glycerin jelly and advised as to the spirit

gum and powder. The theatrical powders are most suitable for the matching of skin shades. Further, spirit gums containing benzene should be avoided. In my experience, Stein's spirit gum is best tolerated.

While certain individuals might question the advisability of plastic surgical procedures in those cases where the supposed disfigurement is congenital or exists only in the vanity of the patient, I think no one would hesitate to advise the use of plastic surgery or a prosthesis in order to remove the disfigurement of accident or disease. The test of the success of a prosthesis is twofold. First, does it relieve the patient and enable him to readjust himself to an ordinary routine of life? and second, is the disfigurement so changed that it ceases to attract universal attention? My experience with patients now wearing gelatin prostheses is sufficient to make me believe the first objective has been attained. Patients often present themselves in a very disturbed state of mind and, frequently, with the history of the loss of weight. In cases where the deformity is due to a malignancy one may wonder whether it is worth while to make a prosthesis. However, I can report one patient who gained 13 pounds in weight within a month's time, and another who gained 19 pounds in weight within six weeks' time following the providing of a suitable prosthesis. This, I think, is sufficient evidence to show that the gelatin prosthesis is satisfactory from the patient's point of view. As to whether or not the eye of the passer-by will accept the prosthesis, — actual observation alone will determine this point. In my experience the prosthesis has proven itself eminently satisfactory. Among other things the gelatin prosthesis possesses a skin-like transparency, is mobile with the features and can be colored to follow seasonal changes in complexion. Photographs indicate the completeness with which the eye is deceived, but it is only by actual observation at conversational distance that a true appreciation of the efficacy of the gelatin prosthesis is evident.

Certain questions arise in the minds of both doctor and patient, which I shall endeavor to answer. Is the prosthesis difficult to make? No, the technique can be learned by anyone of average ability. How long does it take? This material may be prepared and the mold filled in 10 to 15 minutes. This is preferably done at night so that the gelatin may set. The prosthesis is applied and retouched in from 5 to 10 minutes. Does the wearing of the prosthesis harm the skin? An experience over

several years, with the prosthesis attached to scarred surfaces, shows no deterioration of the underlying skin. How often does a prosthesis have to be renewed? Perhaps every day. The average time, I believe, is three days. The prosthesis may be removed, cleaned and replaced so that one may be worn as long as ten days. I have known of a single prosthesis to be worn, without removing, for three weeks.

In conclusion, may I say I feel that in properly selected cases the use of the gelatin prosthesis is, to date, the best method available for those cases not suitable for plastic surgical reconstruction.

DISCUSSION

DR. SAMUEL IGLAUER, Cincinnati: Dr. Batson, as most of us know, has the happy faculty of presenting difficult subjects in a very practical manner. He possesses the unusual combination of a true scientist and a true clinician.

As stated in his paper, facial deformities should, when possible, be restored by plastic surgery, but many considerations mitigate against surgery, such as the time, endurance, and persistence necessary on the part of both the patient and surgeon. Hospitalization is an insurmountable expense in some cases.

The use of gelatin, as demonstrated by the essayist, has certain distinct advantages over more rigid prostheses. As has been demonstrated, the stock gelatin can be colored to match the patient's skin prior to the construction of the prosthesis and requires but slight tinting afterwards.

For the making of the cast, or moulage, upon which the prosthesis is to be modelled, I am of the opinion that Negocoll and Hominit, as described by the late Dr. Poller of Vienna, are most excellent materials, although Dr. Batson seems to prefer plaster of Paris in some cases.

The modelling of the prosthesis requires both technical and sculptural ability.

The method of holding the prosthesis in place with a non-irritating mastic cement is very ingenious.

Providing the patient with a mold enables him to make himself a new nose or a new ear whenever his artificial feature becomes shopworn or soiled. Furthermore, he can, so to speak, keep a "spare" on hand for immediate use when he wishes to attend a party or some other function.

As pointed out by the essayist, a deformed person is entitled to the surgeon's aid, relieving the patient of mental and emotional distress due to his deformity, and at the same time making him a more useful and better adjusted member of society.

Dr. Ferris Smith, Grand Rapids, Mich.: There is little left to be said after the very thorough and excellent presentation of this subject by Dr. Batson and one or two additional ideas by Dr. Iglauer.

I wish to emphasize just two things. One is the economic advantage of this procedure, which was alluded to by Dr. Iglauer, and the other is the fact that no plastic surgeon has yet produced a creditable total ear. It seems highly probable that no plastic surgeon will produce such an ear. At least, if such an ear has been produced, I have never seen any report of it in the literature, nor have I seen any such ear anywhere about the world where I have been accustomed to seeing plastic work.

The very nature of the tissues employed to build an ear preclude the possibility of producing a pleasing one. The third thinnest skin on the body is, probably, the skin covering the ear. If the skin utilized for the reproduction is obtained from an arm, the abdomen or elsewhere on the body, the two layers for the front and posterior covering will produce an ear much thicker than a normal one. If one adds to that an autogenous organic support of sufficient thickness the resultant ear is several times thicker than normal. One is able to obtain quite accurately, quite faithfully, the shape of the helix; to obtain a very creditable lobule to the ear, and to imitate in a rough degree the contours, but when the entire effort is completed, I wish to emphasize again, it is not very creditable.

One can reconstruct half or two-thirds of an ear if the concha remains as a base and produce something which, in my estimation, would be preferable to a prosthetic correction. In my own practice and in the practice of several of my friends, we no longer attempt to produce a total ear. I have two of them still in the making that I would like to refer to any friend who lives at a great distance, and be relieved of ever seeing those patients again.

The second point of great importance is the economic one, alluded to by Dr. Iglauer a moment ago. The plastic construction of an ear requires several operations, several hospitalizations, and expenses which must be borne either by the patient, by society, or by the state. This is not often warranted. I think the same thing applies equally to the reconstruction of a nose. The individual with a champagne appetite, if you wish, and a beer income has no right to expect the state to bear the expense of such a reconstruction, and particularly when it is possible to get such a very pleasing correction as Dr. Batson is able to obtain.

Dr. Francis L. Lederer, Chicago: It is very pleasing to me, after almost ten years of observation, to find someone willing to create an interest in this type of work. He should be complimented on his effort.

As perhaps some of you know, I published a rather extensive article on this subject in the November, 1928, Archives of Otolaryngology, bringing out the technical details which time did not permit Dr. Batson to consider. At that time I brought out some of these economic and technical factors, after observing for a number of years the material in Dr. Beck's clinic, where he finally came to the same conclusion that Dr. Smith voices, namely that these cases very often are love's labor lost.

The technic was developed originally by Warnekros in 1913, and that particular technic involved a material similar to that used in a printer's roller. Perhaps, by way of suggestion, I might say that I believe that the material that Dr. Batson passed around still is too much of that character. It is too soft, too friable.

When I showed my pictures, almost ten years ago, it was very impressive, but there are many disadvantages to this method, despite the fact that it may offer many advantages over surgery — so it isn't as easy as it seems. That psychological factor is not so easily overcome. The immediate adjustment is very fine, and then later on the patient begins to feel that after all this is something artificial.

I have in my files models that are similar to the ones Dr. Batson has, only I got away from this type of model and went to the metallic one, because in a number of instances these were broken and the entire work had to be repeated. I now use brass molds. However, just as the shaving cups remain with the barber, many of these brass molds are still in my possession and the patients are not using them. They apparently do not take the same attitude toward a prosthesis of a nose or an ear that they do toward a dental prosthesis or eye prostheses.

In many instances, there has been dissatisfaction, not with the shape or size of the prosthesis, but the patient still feels that it is unreal. I have the same technic regarding the use of gum mastic for attaching the prosthesis. A patient goes to Florida, and immediately I get a wire, almost an emergency, that he is having difficulty down there; the prosthesis does not stick and the rubber just will not hold its shape. I tried various other measures, like using potassium bichromate to harden this material. I even used formalin, and that made it so hard it became brittle.

Every time the subject comes up — and for ten years I have been corresponding with men over the country who have inquired about it — I never encourage their sending these cases to me, but I ask them to consult someone, particularly a dental technician who might be interested enough to develop the technic. This routine is very difficult. I do not doubt that Dr. Batson can tell you about the difficulties concerning the replacements of the so-called spare parts. It is a technical job and cannot be done by nose and throat men; it is too time-taking.

The prosthesis is a beautiful thing while it lasts and while one's enthusiasm is ardent, but one has to keep encouraging these patients. I have never been able to get that feathery edge to adhere so exactly and stay put. I have the same difficulties that I see on Dr. Batson's ear case, despite the fact that he explained he did not have an opportunity to adjust it properly. Those difficulties still persist and the ear is still artificial. It does replace burdensome surgery, as you have heard, but still there are the difficulties which I had looked forward to having Dr. Batson clarify.

Perhaps we can look forward to a development of this technic. With the interest Dr. Batson has taken and which he will inspire in many others over the country, a better rubber mold and technic is certain to be developed.

I have an ear here that Dr. Beck was kind enough to bring along. It is ten years old and still possesses that same elasticity that the material

had at that time when I brought the method to the attention of American specialists.

I feel, just as you gentlemen do, that we need a substitute for this surgery that is apparently unavailing, and yet this method as presented in itself needs considerably more development than has been shown up to the present day.

I give particular credit to a technician in the late Passow's laboratory in Berlin by the name of Klocke, who is a very ingenious fellow. This type of man is in a better position certainly than we are as physicians to develop these things. He spends considerable time with it.

Dr. Joseph C. Beck, Chicago: This presentation is certainly of great importance and will be welcomed, even though it may not be entirely original, by many individuals who are candidates for reconstructive or plastic surgery. After all, as Dr. V. Blair said, "What we do in many plastic operations is transform a patient from a deformity to a monstrosity."

That has been my experience in a great many cases in doing reconstructive work in noses, and certainly in ears, as Dr. Ferris Smith has told you, it has also been his. No matter whether the deformity is from disease, from an operation, a war injury or an automobile injury, the marked destruction and deficiency of skin and underlying tissues make difficulties for operations.

From Dr. Lederer's discussion — which is absolutely correct, he having been given the work to do by me at the time this matter of artificial nose making came to our attention. To put this in application — I gathered a little too much of a doubt as to his opinion of the value of this method of correction.

If this is not the best material that Dr. Batson presents today, it surely will be improved upon. I came here with a great deal of anticipation as to what I would learn from Dr. Batson's presentation, and I am not disappointed. I am sure it is going to be better in the future than what we have had presented today.

It is true that a patient does not like artificial noses and ears. Many of them refuse to wear them. I want to present a case here, a plaster cast of a woman who had had several extensive radical operations of the frontal sinus, and one of the most extensive sizes of the frontal sinus. It went posteriorly almost to the back of the apex of the orbit and externally or laterally to the temporal region and high up, above the hair line. The end result was a complete destruction of the frontal bone. The woman is sixty-eight years old now. To begin with, she refuses any kind of plastic reconstruction, and it would be very difficult, the way the recesses are formed, to try to put an anterior surface of bone or skin flaps in this case. This material shown by Dr. Batson is going to serve, I believe, an excellent purpose in this particular individual.

These molds are of great importance in cases of resection of the upper jaw for malignancy, while we are waiting for plastic work, and we must wait, else we may have covered up recurrence. Dressings and all sorts of prostheses are really very poor substitutes unless this material

demonstrated here today might be brought into use. Repeating: That is a very important thing for prostheses while waiting for definite knowledge that there is no recurrence from malignancy.

DR. OSCAR V. BATSON, closing: I appreciate the extended discussion and I want to thank those who participated, Dr. Iglauer, Dr. Ferris Smith, Dr. Lederer, and Dr. Beck. I also wish to acknowledge my indebtedness to Dr. Iglauer for preparing and sending to me the casts of these two patients from his clinic.

The replaceable prosthesis, as Dr. Lederer has told you, is a very old idea. I do not know its exact genesis, but certainly the Hennings, of Vienna, were quite familiar with it. The method has been published in several languages by different authors, but many times the formulæ that have been given do not give satisfactory results, either because of the indefiniteness of the formula or because of the lack of availability of the material. In this connection I should like to add that I see no virtue in the dichromates added to various formulæ other than that they give a yellow color to the mass.

I think that gelatin, not glue, is the ideal material, although for very warm weather, some of the high grade, rather transparent glues work very well. I have prepared such material for a patient for summer use in South Carolina. The mass which I have shown here contains 60 per cent glycerin and 40 per cent gelatin, and it has not seasoned quite long enough to lose all of its water. It is a bit too soft as yet for use by the patient.

There has been available recently a synthetic rubber which can be melted and poured, but I do not believe that this material will be as satisfactory as gelatin except for replacements about the maxilla, where the prosthesis is being constantly bathed in fluids.

I am familiar with metal molds but I prefer to use dental stone. This may well be a personal matter. A swaged mold is not satisfactory because the surface is too smooth to imitate skin. The cast metal mold is necessary to produce the required irregularities.

If the patient is not interested in his personal appearance, a replaceable prosthesis should not be used. It is largely true, as Dr. Lederer suggested, that you never finish with a prosthesis case. Difficulties do arise and you have to aid the patient in taking care of them. In retrospect, however, I think that you will conclude that the appreciation of the patient for being returned to normal activity compensates for the time you have spent.

RESEARCH REPORT 1935

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AN EXPERIMENTAL INVESTIGATION OF TINNITUS*

W. J. McNALLY, M.D.
E. A. STUART, M.D.; T. F. REID, M.D.; L. H. McCONNELL, M.D.

MONTREAL

THIS PRELIMINARY REPORT is mainly concerned with the study of nineteen patients suffering from tinnitus. Care was taken not to include any case of objective tinnitus. There was no selection or classification made as to the age of the patient, as to the kind of noise heard, as to its duration, as to its being unilateral or bilateral, as to the type of ear disease present nor as to the probable etiology of the head-noise. Some of the patients complained more of tinnitus, but others complained more of deafness.

The investigation was undertaken as the result of an idea derived from reading Dr. Albert A. Gray's article "The Otosclerosis Problem" (1934). Part of Dr. Gray's conclusions may be summarized as follows: The essential causative factor in otosclerosis is a gradually increasing defect of the vasomotor mechanism which governs the nutrition of the structure of the organ of hearing as a whole. Failure of this reflex results in a deficient blood supply. It is stressed that the lesions in the nerve tissue in otosclerosis are just as important as those in the bony tissue and that the pathological changes in the bone bear no relationship to the severity of the tinnitus. The tinnitus is due to a lesion in the auditory nerve tracts either in the ear or in the brain. He refers to the temporary spontaneous improvement in hearing which may take place in otosclerosis and considers it to be due to a reflex act in the vasomotor system in general, bringing about an increased cerebral circulation. In some

* Research authorized by the Council and financed by the American Academy of Ophthalmology and Otolaryngology, from the Department of Otolaryngology of the Royal Victoria Hospital, Montreal, Canada.

patients he found that a similar improvement followed the inhalation of amyl nitrite.

Dr. Gray does not mention what happens to the tinnitus in those cases of otosclerosis with sudden temporary return of hearing supposedly due to increased circulation from a general vasomotor reflex. If the cause given for the temporary return of hearing is correct, an attempt at interference with the cerebral vasomotor mechanism is a reasonable approach to the study of tinnitus since it is so closely allied to the deafness of otosclerosis. For a complete description of tinnitus the reader is referred to Gower's "Diseases of the Nervous System" (1888). He describes the varying character of the noises, the etiology, the differential diagnosis, the treatment and the prognosis. There is very little to change of what was said 47 years ago. No attempt shall be made at a general discussion of the different theories about tinnitus to be found in the literature. We shall refer only to those papers which are thought to have a direct bearing upon our experiments.

INCIDENCE OF TINNITUS

The incidence of tinnitus in ear and intracranial disorders is important when considering its possible origin. Fowler (1912) reviewed 2,000 consecutive ear cases and found tinnitus present in two-thirds of the cases. In his series, tinnitus was present in 67% of catarrhal non-suppurative disease of the ear. Fraser (1932) states that if severe tinnitus is present in a case of chronic catarrh of the middle ear, secondary otosclerosis should be suspected. In chronic suppurative otitis media, Fraser (1932) finds that tinnitus is an occasional symptom, but it is rarely well marked. Fowler gives the percentage as 33. In otosclerosis, Gray (1934) finds tinnitus rarely absent. In the review of the literature on otosclerosis up to 1928 carried out by a committee of the American Otological Society (Duel 1929), it is seen that tinnitus is considered to be a most troublesome and one of the most common symptoms in otosclerosis. The percentage is variably given as from 75 to 90. In a series of 42 cases of Ménière's syndrome complex, Dandy (1934) finds tinnitus present in all but one case. Crowe, Guild and Polvogt (1934) report that tinnitus was not a prominent symptom in a series of 79 cases of high-tone deafness, in 75% of which a lesion was found microscopically in the basal turn of the cochlea. These are proven cases of nerve deafness, and tinnitus was present in 21 cases,

but they add, "it was not the distressing symptom so commonly observed in patients with otosclerosis or other lesions of the conductive mechanism."

In order to determine the incidence of tinnitus in intracranial disease, permission was obtained from Dr. Wilder Penfield to make a survey from the records of the neurosurgical service of the Royal Victoria Hospital, Montreal. Three hundred and fifty-one of the likely case reports were searched for the presence of tinnitus. (See Table 1).

TABLE 1 — *Tinnitus in Intracranial Disease*

TYPE OF CASE	NO. OF CASES REVIEWED	TINNITUS PRESENT IN	EVIDENCE OF LOCAL EAR DISEASE IN	TINNITUS WITHOUT LOCAL EAR DISEASE
Epilepsy	245	22	5	17
Frontal Lobe Tumors	33	6	3	3
Parietal Lobe Tumors	21	2	..	2
Temporal Lobe Tumors	17	3	1	2
Cerebellar Tumors	30	10	3	7
Eighth Nerve Tumors	5	3	..	3
	351	46	12	34

It must be remembered that tinnitus was not paramount in the minds of the men who wrote the case reports, so that it is just possible that a mild tinnitus may have been overlooked in the presence of more urgent or more troublesome symptoms. On the other hand, these case reports are exceptionally complete and I am sure that the percentage of error from this source is extremely small.

When tinnitus was noted in a case, evidence of local ear disease was sought in the case report which might serve as an adequate explanation of the tinnitus in the absence of the intracranial lesion. The distribution of 12 such cases is given in Table 1 under the column of local ear disease.

Where the noise is in the nature of a definite aura of epilepsy it is not included as a case of tinnitus. There were 12 such cases in the 245 cases of epilepsy. When there is an aura of sound and the localization of the origin of the epileptic seizure has been proven at operation, it has always been in the temporal lobe. In only a few cases has Dr. Penfield been able to reproduce sound sensation by stimulation of the temporal lobe cortex

in patients under local anesthesia. The patient then hears a drumming sound (see Penfield and Gage 1933 and Penfield 1935).

This brief summary of the incidence of tinnitus would indicate that tinnitus is most common and most troublesome in otosclerosis and Ménière's syndrome.

ETIOLOGY

Innumerable theories have been advanced ascribing the etiology of tinnitus to some condition in or about the middle ear. We are not in a position to discuss these theories.

Fraser (1929) considers the etiology to be a neuritis of the cochlea (spiral ganglion cells), and Gray (1934) feels that it is the nerve lesion in otosclerosis which is responsible for the tinnitus. On the other hand in the proven nerve-deafness cases of Crowe and his co-workers, though tinnitus is present in 25% of the patients, it is never a prominent symptom. Dandy found that after section of the eighth nerve for Ménière's syndrome, that the tinnitus persisted in 50% of the cases even though the condition was unilateral in all but six of the forty-two patients. This would place the lesion causing tinnitus proximal to the point of section of the nerve. In 351 cases of intracranial disease we find tinnitus present in 34 patients in which there is no gross evidence of middle ear disease, and it is most common when the lesion is within the posterior fossa.

Leaving out of consideration tinnitus which may be caused by some condition in or about the middle ear, it is evident that there are some cases of tinnitus which are due to a lesion of the nervous mechanism of the ear and brain.

CLINICAL EXAMINATION

In each of the 19 cases here recorded a careful history was taken of the local condition and of the general health. Deafness and tinnitus were traced as completely as possible, with any factors which appeared to influence them. The family history was searched. The following points were inquired for in detail: ear disease; nose and throat disturbances; severe general illnesses and their nature; war injuries or other accidents; use of tea, coffee, alcohol and tobacco; use of medicines as routine tonics or purgatives, etc.; nature of occupation; and finally the diversions, such as shooting, etc.

Case and Name	Age and Sex	History of Deafness and Tinnitus	Family History	General Medical History and Examination	Nose and Throat Examination	Ear Drums		Audiometer Examination					
								Total Loss per Cent		Location of Max. Loss		A.B.C. in Sensation Units 512 D.V.	
						Right	Left	Rt.	Lt.	Rt.	Lt.	Rt.	Lt.
1. A.P.	28 F.	Onset of deafness insidious at puberty 14 yrs. ago. Worse after childbirth, 4 yrs. ago, when tinnitus began in both ears.	Irrelevant	Vasomotor rhinitis 3 yrs. Pelvic inflammatory disease 2 yrs.	Periodically nasal mucosa swollen, watery. Nasal discharge, otherwise, negative.	Normal	Normal	31	31	Abrupt High	Abrupt High	65	75
2. A.D.	36 F.	Deafness insidious at onset 8 yrs. ago. Worse in cold weather. Tinnitus in left ear began 1 yr. ago.	1 sister deaf for yrs. Details unknown	Normal	Normal	Dull	Dull	23	32	Uniform	Uniform	65	65
3. L.G.	44 M.	Deafness and tinnitus insidious; onset 15 yrs. ago.	Irrelevant	Normal	Normal	Dull	Dull	47	58	Gradual High	Gradual High	70	70
4. E.T.	33 F.	Deafness and noise in both ears for many years. Onset indefinite. Head injury at age of 3 yrs.	Irrelevant	Spontaneous vert. nystagmus with divergent strabismus left eye; cause unknown. Menorrhagia. Renaud's dis.	Normal	Retracted	Retracted	42	28	Uniform	Uniform	75	75
5. P.R.	37 F.	Tinnitus and slight deafness in left ear for 26 yrs. following scarlet fever. Tinnitus in right ear 1 mo.	Irrelevant	Normal	Normal	Normal	Retracted	21	32	Uniform	Gradual High	70	70
6. H.L.	35 M.	Noise and deafness in left ear 4 mos. Discharge from left ear 3 mos.	Irrelevant	Pulmonary tuberculosis 8 yrs. Fibroid type in each apex.	Normal	Normal	P'forated and disch'ging	19	46		Gradual High	70	70
7. D.B.	25 F.	Deafness and tinnitus following pneumonia 12 yrs. ago. Tinnitus in right ear only.	Irrelevant	Chronic menorrhagia and Renaud's disease.	Normal	Normal	Normal	47	59	Gradual High	Gradual High	70	Not Heard
8. V.O.	19 F.	Deafness and tinnitus with insidious onset at 11 yrs. of age. C.S.O.M. in childhood. Tinnitus in right ear only.	Irrelevant	Normal	Normal	Scarred	Normal	48	48	Uniform	Uniform	50	50
9. A.P.	36 F.	Buzzing in left ear started insidiously 4 yrs. ago.	Irrelevant	Normal	Recent acute maxillary sinusitis now cured.	Normal	Normal	14	21	Uniform	Uniform	80	75
10. J.P.	49 M.	Progressive deafness in left ear following typhoid fever 5 yrs. ago. Tinnitus in left ear 2 mos.	Father deaf. Details unknown.	Epileptiform seizures. Treated lues 19 yrs. ago. Bl'd Was'm'n 3+ now.	Normal	Dull	Dull	23	50		Gradual High	45	50
11. G.C.	47 M.	Deafness 4 yrs., first in left ear, then in right. Tinnitus for 3 yrs. in left ear.	Irrelevant	Normal	Normal	Thickened	Thickened	Hears but few notes in each ear in midrange at max. intensity.				90	95
12. H.C.	41 F.	Tinnitus in right ear, insidious onset 10 yrs. ago. Never conscious of deafness. Pain in left ear 2 mos.	Irrelevant	Hypochromic anæmia & possibly nervus intermedius neuralgia, left.	Normal	Normal	Normal	30	20	Uniform	Uniform	65	65
13. A.M.	42 F.	C.S.O.M. 7 yrs. in right ear. Deafness, right ear, with tinnitus progressive for 2 yrs.	Irrelevant	Cholecystotomy 1934	Normal	Dry Perforation	Scarred	54	46	Gradual High	Gradual High	70	70
14. M.S.	42 F.	Deafness 7 yrs. in both ears. Tinnitus 4 yrs. Worse in cold weather.	Irrelevant	Normal	Chronic septic tonsillitis.	Dull	Dull	59	62	High & Low	High & Low	80	100
15. W.H.	42 M.	Gradual onset of deafness since age of 15 yrs. in right ear. Deafness in left ear 6 yrs. ago. Tinnitus in left ear 3 yrs. ago.	1 brother, has otoscler. 1 sister deaf.	Mild tuberculosis n right apex of lung.	Normal	Dull	Dull	72	75	High & Low	Gradual High	80	85
16. P.P.	39 M.	Noise in left ear 15 yrs. following the firing of a gun.	Irrelevant	Normal	Hypertrophied tonsils.	Normal	Normal	21	29		Abrupt High	60	70
17. H.P.	52 F.	Noise and dizziness, left ear, for 8 months.	Irrelevant	Kidney and liver disease. Ménière's syn.	Normal	Normal	Normal	19	41		Uniform	65	65
18. L.A.	39 F.	Progressive buzzing in right ear since Sept., 1934, not relieved by Politzerization. Duration of deafness unknown.	Irrelevant	Normal	Normal	Normal	Normal	49	30	Uniform	Uniform		
19. E.L.	33 F.	Gradual onset of deafness and tinnitus at age of 16 yrs. made worse by a miscarriage and pregnancy.	Irrelevant	Normal	Normal	Normal	Normal	41	59	Gradual High	Gradual High		

NOTE — A.B.C. denotes Absolute Bon

TABLE 2 — *Synopsis of Nineteen Cases of Tinnitus Aurium*

	Ear Drums		Audiometer Examination										Weber Referred	Schwabach in Secs.		Monochord Upper Tone Limit in D.V.		Diagnosis	Case No.	Amyl Nitrite	Deep Breathing
			Total Loss Per Cent		Location of Max. Loss		A.B.C. in Sensation Units 512 D.V.		Whispered Voice		Rinne										
	Right	Left	Rt.	Lt.	Rt.	Lt.	Rt.	Lt.	Rt.	Lt.	Rt.	Lt.		Rt.	Lt.	Rt.	Lt.				
...al mu...watery. ...rge, ...gative.	Normal	Normal	31	31	Abrupt High	Abrupt High	65	75	5 ft.	5 ft.	+	+	Left	— 8	— 5	12,000	11,000	Otosclerosis and nerve deafness.	1.	N.C.	N.C.
	Dull	Dull	23	32	Uniform	Uniform	65	65	8 ft.	8 ft.	+	+	Left	Normal	— 5	14,000	12,000	Middle ear and nerve deafness.	2.		N.C.
	Dull	Dull	47	58	Gradual High	Gradual High	70	70	2 ft.	At Ear	—	—	Not Ref.	Normal	— 2	15,000	8,000	Otosclerosis and nerve deafness.	3.	N.C.	N.C.
	Retracted	Retracted	42	28	Uniform	Uniform	75	75	1 ft.	2 ft.	+	+	Not Ref.	—20	—20			Traumatic nerve deafness.	4.	Worse	Worse
	Normal	Retracted	21	32	Uniform	Gradual High	70	70	6½ ft.	½ ft.	+	+	Left	—10	—10	12,000	13,000	Post scarletinal nerve deafness. Otosclerosis?	5.	N.C.	
	Normal	P'forated and disch'ging	19	46		Gradual High	70	70	5 ft.	At Ear	+	+	Left	—12	— 7			Middle ear and toxic nerve deafness, left. (Tbc?)	6.		
	Normal	Normal	47	59	Gradual High	Gradual High	70	Not Heard	Not Heard	Not Heard	+	+	Not Ref.	Normal	—20	15,000	4,000	Toxic nerve deafness (Post-pneumonic).	7.	N.C.	N.C.
	Scarred	Normal	48	48	Uniform	Uniform	50	50	At Ear	At Ear	—	—	Not Ref.	+10	+10	17,000	17,000	Otosclerosis.	8.	Worse	Worse
maxil-now	Normal	Normal	14	21	Uniform	Uniform	80	75	15 ft.	15 ft.	+	+	Not Ref.	— 5	—10	17,000	15,000	Early nerve deafness.	9.		Worse
	Dull	Dull	23	50		Gradual High	45	50	3 ft.	Not Heard	—	—	Right	+20	+12	14,000	10,000	Nerve deafness left. (Typhoid or lues).	10.	Worse	Worse
	Th'ck-ened	Thick-ened	Hears but few notes in each ear in midrange at max. intensity.				90	95	Loud Shout	Noth-ing			Not Ref.					Nerve deafness.	11.	Worse	N.C.
	Normal	Normal	30	20	Uniform	Uniform	65	65	5 ft.	5 ft.	+	+	Not Ref.	— 5	— 5			Early nerve deafness, toxic?	12.		Worse
	Dry Perforation	Scarred	54	46	Gradual High	Gradual High	70	70	At Ear	1 ft.	+	+	Right	—10	—15			Middle ear deafness and nerve deafness.	13.		Worse
ptic	Dull	Dull	59	62	High & Low	High & Low	80	100	At Ear	At Ear	—	—	Right	—15	—12			Chronic catarrhal otitis media and nerve deafness.	14.	Improved	N.C.
	Dull	Dull	72	75	High & Low	Gradual High	80	85	Not Heard	Not Heard	—	—	Right	—20	— 8	Not Heard	Not Heard	Otosclerosis and advanced nerve deaf.	15.	Improved	
hiel	Normal	Normal	21	29		Abrupt High	60	70	12 ft.	8 ft.	+	+	Not Ref.	Normal	— 8	15,000	12,000	Traumatic nerve deafness? Left.	16.		
	Normal	Normal	19	41		Uniform	65	65	8 ft.	½ ft.	+	+	Right	—10	—24			Nerve deafness and Ménière's syndrome.	17.		N.C.
	Normal	Normal	49	30	Uniform	Uniform			5 in.	15 in.	—	—	Right	Normal	Normal			Middle ear deafness.	18.		
	Normal	Normal	41	59	Gradual High	Gradual High			At Ear	Nil	+	+	Not Ref.					Otosclerosis.	19.	N.C.	Improved

NOTE — A.B.C. denotes Absolute Bone Conduction.

Deep Breathing	Spheno-palatine Ganglion Injection	Sodium-free Diet	Intravenous Glucose Injection	Constricting Neckband	Ephedrine		Bellafoline		Ergotamine		Ergotamine followed by Mecholyl Hypodermic		Bellafoline followed by Ephedrine Hypodermic		Case No.
					By Mouth	Hypodermic	By Mouth	Hypodermic	By Mouth	Hypodermic	After Ergotamine	After Mecholyl	After Bellafoline	After Ephedrine	
N.C.	N.C.	N.C.	N.C.	N.C.	Worse	N.C.	Worse	Improved	Worse	N.C.	N.C.	Improved	N.C.	Improved	1.
N.C.	N.C.	N.C.		Worse		Worse	Improved	Worse	N.C.	Improved	N.C.	N.C.	N.C.	N.C.	2.
N.C.	N.C.	Improved	Worse	N.C.		Improved	Improved	Improved	Worse	N.C.	N.C.	N.C.	Improved	N.C.	3.
Worse	N.C.	N.C.	N.C.	Worse		N.C.	N.C.	N.C.	Worse	N.C.			N.C.	N.C.	4.
	Improved	Not Tolerated	N.C.				Improved	Worse	Worse	Worse					5.
	N.C.					N.C.	Improved	Improved		N.C.	Not carried out on advice of medical clinic because of tuberculosis.				6.
N.C.	N.C.	Improved	Improved	N.C.		Improved		N.C.	N.C.	N.C.	N.C.	N.C.	Improved	Much Improved	7.
Worse	N.C.	N.C.	N.C.	N.C.	Improved	Improved	Improved	Improved	Improved	N.C.	N.C.	Improved	Improved	Improved	8.
Worse	N.C.	Not Tolerated	Improved	N.C.		N.C.	N.C.	Improved		N.C.	N.C.	N.C.	N.C.	Worse	9.
Worse	N.C.	Discontinued	N.C.	N.C.		N.C.	Improved	N.C.	Worse	Worse	Worse	N.C.	N.C.	Worse	10.
N.C.	Improved	N.C.				N.C.	Improved	N.C.	N.C.	N.C.	N.C.	N.C.			11.
Worse					Improved	Improved	N.C.	N.C.		N.C.	N.C.	Worse	Improved	Much Improved	12.
Worse				N.C.		Worse	N.C.	N.C.		N.C.	N.C.	N.C.	Improved	N.C.	13.
N.C.		N.C.		N.C.	Improved	Improved		N.C.	N.C.	Improved	N.C.	N.C.			14.
				N.C.	Improved	Improved	Improved	N.C.	Improved	N.C.	Worse	Improved	N.C.	Improved	15.
	N.C.	Not Tolerated	N.C.	Worse		N.C.		N.C.		Worse					16.
N.C.								N.C.							17.
	N.C.	Worse	N.C.			N.C.			N.C.						18.
Improved						N.C.				Worse					19.

NOTE — N.C. denotes No Change.

HENO- LATINE IGLION ECTION	SODIUM- FREE DIET	INTRA- VENOUS GLUCOSE INJECTION	CON- STRICT- ING NECKBAND	EPHEDRINE		BELLAFOLINE	
				BY MOUTH	HYPODERMIC	BY MOUTH	HYPODERMIC
N.C.	N.C.	N.C.	N.C.	Worse	N.C.	Worse	Im- proved
N.C.	N.C.		Worse		Worse	Im- proved	Worse
N.C.	Im- proved	Worse	N.C.		Im- proved	Im- proved	Im- proved
N.C.	N.C.	N.C.	Worse		N.C.	N.C.	N.C.
Im- roved	Not Tol- erated	N.C.				Im- proved	Worse
N.C.					N.C.	Im- proved	Im- proved
N.C.	Im- proved	Im- proved	N.C.		Im- proved		N.C.
N.C.	N.C.	N.C.	N.C.	Im- proved	Im- proved	Im- proved	Im- proved
N.C.	Not Tol- erated	Im- proved	N.C.		N.C.	N.C.	Im- proved
N.C.	Discon- tinued	N.C.	N.C.		N.C.	Im- proved	N.C.
Im- oved	N.C.				N.C.	Im- proved	N.C.
				Im- proved	Im- proved	N.C.	N.C.
			N.C.		Worse	N.C.	N.C.
	N.C.		N.C.	Im- proved	Im- proved		N.C.
			N.C.	Im- proved	Im- proved	Im- proved	N.C.
N.C.	Not Tol- erated	N.C.	Worse		N.C.		N.C.
							N.C.
N.C.	Worse	N.C.			N.C.		
					N.C.		

NOTE — N.C. denotes No Change.

DERMIC	ERGOTAMINE		ERGOTAMINE FOLLOWED BY MECHOLYL HYPODERMIC		BELLAFOLINE FOLLOWED BY EPHEDRINE HYPODERMIC		CASE NO.
	BY MOUTH	HYPODERMIC	AFTER ERGOTAMINE	AFTER MECHOLYL	AFTER BELLAFOLINE	AFTER EPHEDRINE	
Im-oved	Worse	N.C.	N.C.	Im-proved	N.C.	Im-proved	1.
Worse	N.C.	Im-proved	N.C.	N.C.	N.C.	N.C.	2.
Im-oved	Worse	N.C.	N.C.	N.C.	Im-proved	N.C.	3.
N.C.	Worse	N.C.			N.C.	N.C.	4.
Worse	Worse	Worse					5.
Im-oved		N.C.	Not carried out on advice of medical clinic because of tuberculosis.				6.
N.C.	N.C.	N.C.	N.C.	N.C.	Im-proved	Much Im-proved	7.
Im-oved	Im-proved	N.C.	N.C.	Im-proved	Im-proved	Im-proved	8.
Im-oved		N.C.	N.C.	N.C.	N.C.	Worse	9.
N.C.	Worse	Worse	Worse	N.C.	N.C.	Worse	10.
N.C.	N.C.	N.C.	N.C.	N.C.			11.
N.C.		N.C.	N.C.	Worse	Im-proved	Much Im-proved	12.
N.C.		N.C.	N.C.	N.C.	Im-proved	N.C.	13.
N.C.	N.C.	Im-proved	N.C.	N.C.			14.
N.C.	Im-proved	N.C.	Worse	Im-proved	N.C.	Im-proved	15.
N.C.		Worse					16.
N.C.							17.
	N.C.						18.
		Worse					19.

UNIV. OF MICH.

A complete general medical examination was carried out by the medical clinic of the hospital and where necessary blood chemistry and other special examinations were done. The blood Wassermann was done as a routine. It was negative in all but Case 10. This patient was receiving antiluetic treatment.

Examination of the hearing was carefully done in order to render the diagnosis of the local ear condition as exact as possible. To determine the condition of the vestibular mechanism, the caloric test was done in each case and the rotation tests were carried out in some patients. The results were normal in all but one. Case 14 showed a diminished response but the reactions were still present.

Each patient was Politzerized or the eustachian tube was catheterized, but Cases 2 and 12 were the only ones to show temporary improvement of the tinnitus.

It was decided to include a summary of the examination and treatment in the form of a chart, Table 2, instead of recording the individual case reports.

PROCEDURES FOLLOWED

The various procedures which were carried out in the hope of influencing the tinnitus, either to lessen or to increase it, will be discussed in the order in which they appear on the summary chart (Table 2). The reason why each procedure was chosen will be given.

Amyl Nitrite—Gray (1934) states that following the inhalation of amyl nitrite some patients with otosclerosis experienced a temporary improvement in hearing due to a general redistribution of the blood from a vasomotor reflex, the ear receiving a temporarily increased blood supply. We wished to observe the effect of such changes upon the tinnitus, especially the effect of lowered blood pressure, since Wolff (1929) found that inhalation of amyl nitrite in cats caused a definite fall in arterial blood pressure, a dilatation of the pial blood vessels and a rise in cerebrospinal fluid pressure.

The patients were placed in the prone position and after a period of rest, the blood pressure was recorded. A 3 minim ampoule of amyl nitrite wrapped in gauze was held close to the nose and mouth. The blood pressure was recorded at two minute intervals till the general effects of the drug had passed. If necessary, the procedure was repeated. In every case a

marked flushing of the face and arms occurred and lasted about ten minutes. The patient was asked repeatedly if any change was noted in the head-noise or the hearing. The hearing was not tested during the experiment. It is noteworthy that a preliminary rise of blood pressure occurred in every case and lasted for about five minutes. This was unexpected. In the eleven patients tested, the noise was not affected in five; four were made slightly worse and in two the noise was improved. There was no special change in hearing noted by any of these patients. The rise of blood pressure following the inhalation of amyl nitrite was always greater than the subsequent fall (see Table 3).

TABLE 3 — *The Effect of Inhalation of Amyl Nitrite on Systemic Blood Pressure*

CASE NO.	RESTING B. P. S.D.	HIGHEST READING (ABOUT 5 MINUTES AFTER INHALATION) S.D.	LOWEST READING (ABOUT 10 MINUTES AFTER INHALATION) S.D.
1	100/70	118/90	100/65
3	120/78	145/90	115/80
4	115/85	140/90	110/80
5	110/70	140/70	110/
7	120/90		
8	150/80	170/	150/
10	110/75	120/80	105/80
11	120/70	137/75	104/65
14	104/60	118/	90/55
15	142/80	170/95	118/80
19	130/90	120/90	160/90

Hyperventilation (Deep Breathing) — Hyperventilation has been used in epileptics to induce a convulsion in order that the seizure may be studied (Gibbs, Lennox and Gibbs 1934). The rapid continuous breathing causes a blowing off of the CO_2 and an alkalosis of the blood results. There is a sharp decline in blood flow and a slight fall in blood pressure. The epileptic seizures that were produced occurred two minutes after the lowest point in blood flow was reached. The above authors felt that vasoconstriction and anæmia played a part.

It was felt that if deep breathing caused a change in cerebral circulation that some effect might be shown upon tinnitus. The patients were required to breathe deeply and rapidly for five minutes, three times a day. They were asked to make a note

of the effect, if any, upon the head-noise and to present this diary at the clinic the following week. One patient, Case 17, was made quite ill by the deep breathing. Case 12 states that the noise in her left ear began for the first time after the deep breathing exercise. The test was carried out in fourteen patients. There was no change in six; six were made worse, and in two the tinnitus was improved.

Spheno-Palatine Ganglion Injection — Ruskin (1925) advocates painting the spheno-palatine ganglion region as a possible cure for tinnitus. He describes a case relieved by cocainization. With this in mind we chose several cases with unilateral tinnitus for injection of the homolateral spheno-palatine ganglion region with novocaine, one-half per cent. One cc. of the solution was injected through the pterygo-palatine canal using a needle designed by Campbell (1929). The needle is inserted to a depth of 2½ to 3 centimeters from the surface of the mucous membrane of the hard palate. There is usually severe pain during the injection. The pain is on the side of the injection and chiefly deep in the cheek, but it radiates over that side of the head. The success of the injection is determined by anesthetization of the posterior pharyngeal pillar on the same side. The patients were questioned frequently during the subsequent hour as to the effect of the injection upon the tinnitus and they were asked to note any change in the tinnitus during the next twelve hours. Thirteen cases were injected. Eleven cases were not affected; in one case the noise was worse and in one case the noise was completely gone for fifteen minutes, about ten minutes after the injection.

Sodium-Free Diet — Furstenberg, Lashmet and Lathrop (1934) reported the results of a very thorough investigation of six cases of Ménière's syndrome in which the metabolism of water and sodium were carefully controlled. They found that the symptoms of Ménière's syndrome are due to the retention of sodium by the body. By permitting as small an intake of sodium as possible in the diet and by preventing accumulation of sodium in the body by the administration of ammonium chloride, they were able to keep these patients free from attacks. On the other hand, by administration of sodium they were able to precipitate an attack. The authors do not mention specifically what happened to the tinnitus in these cases, but we decided to try this régime in several cases of tinnitus even in the ab-

sence of Ménière's syndrome, since tinnitus is such an invariable accompaniment of this latter disease. The routine followed in our cases was to instruct the patient in the necessity of avoiding sodium in any form. They were given a copy of a sodium-free diet very kindly supplied by Dr. Furstenberg. They were given 45 grains of ammonium chloride three times a day with each meal. This was to be taken for three consecutive days, then miss two days and then repeat till the end of the test. The ammonium chloride was eventually given in pill form, several other methods of administration having first been attempted. The patients in this series were kept on this routine for two weeks. Other cases not included in this group were kept on the routine for four or five weeks. In many of the cases the hydrogen ion concentration and the carbon dioxide combining power of the blood were examined before and after the treatment (see Table 4).

TABLE 4 — *Blood Examination to Determine Hydrogen Ion Concentration and Carbon Dioxide Combining Power, Before and After Sodium-Free Diet*

CASE	BEFORE		AFTER	
	*p*H	CO_2 VOLS. %	*p*H	CO_2 VOLS. %
1	7.21	66.2	7.19	43.7
2	7.21	66.2	7.36	62.2
3	7.32	68.3	7.30	63.5
4	7.21	68.1	7.33	56.7
5	7.21	64.3		
7	7.32	63.9	7.23	59.3
8	7.32	69.5	7.36	55.6
9	7.31	66.3		
10	7.31	62.5		
11	7.31	67.3	7.27	73.0
14	7.20	59.6	7.31	64.4
16	7.28	71.0		
18	7.23	63.5	7.30	64.5

Since the diet and the ammonium chloride tend to produce an acidosis, it should be noted that there is very little lowering of the *p*H and CO_2, in fact there has been a rise shown by some of the patients (see Table 4). This may explain why there were no definite improvements shown by this group of cases. It should be mentioned, however, that these cases were on home

treatment and would follow the treatment as well, or as badly, as the average case of Ménière's syndrome on the same treatment, and yet in the last mentioned disease relief from vertigo has been reported. Thirteen patients were started on the treatment but only nine carried out the régime for the two weeks' trial period. It was not tolerated by four. Six of the patients experienced no change in the tinnitus; the tinnitus was worse in one case and it was improved in two cases. The one case in this series which is diagnosed as Ménière's syndrome was admitted to hospital and put on the diet and treatment, but she would not cooperate. She left the hospital and would not complete the treatment at home. From these cases and from other patients suffering from tinnitus who have been put on the above régime, we have been forced to conclude that the ordinary tinnitus case is not benefited. We have not sufficient experience to say what the effect is upon tinnitus of definite Ménière's syndrome.

Intravenous Glucose Injection — One patient reported relief of the head-noise after eating honey. She was asked to eat large quantities of honey and several other patients were asked to do likewise. There was no definite improvement. It was thought that the glucose in the honey may have been a factor causing dehydration. For this reason, eleven cases were asked to take six ounces of glucose a day in the form of corn syrup and to take magnesium sulphate, one drachm, three times a day. There was no definite effect upon the tinnitus. This régime was probably quite inadequate to produce any dehydration. It was decided to resort to more acute methods of dehydration.

Portman (1927) suggests that Ménière's syndrome is due to increased pressure in the saccus endolymphaticus and membranous labyrinth, either from pathologic changes adjacent to the saccus or secondly, from endolabyrinthine causes and "auricular glaucoma." He describes an operation for puncture of the saccus which cures the vertigo but he makes no mention of the tinnitus even though he speaks of Ménière's triad. On the basis of this increased endolymphatic pressure theory, Felderman and Dyson (1929) describe a method which they state lowers intralabyrinthine pressure by promoting osmosis from the saccus endolymphaticus into the subarachnoid space. (It must be pointed out that the saccus endolymphaticus does not communicate with the subarachnoid space.) By withdrawing 5 cc. of spinal fluid and by inserting 4 cc. of 5 per cent glucose solution through

the lumbar puncture needle, they lower the cerebrospinal fluid pressure. In a series of five patients suffering from tinnitus which they studied for five weeks, they obtained relief in three cases.

Hughson (1932) has described animal experiments in which he simultaneously took readings of intracranial and intralabyrinthine pressure and at the same time made electrical records of the animals' hearing while the cerebrospinal fluid pressure was altered by intravenous injections of hypo- and hypertonic solutions. As a hypotonic solution 50 cc. of distilled water was injected intravenously. The cerebrospinal fluid rose, followed by a rise in intralabyrinthine pressure. There is a lag of the labyrinthine pressure rise at the beginning, but it continues to go up for a short while after cerebrospinal fluid pressure has begun to recede, probably due to the narrowness of the aqueductus cochlea. There is a slight tendency towards improvement of sound transmission through the ear. When a hypertonic solution, 9 cc. of 30 per cent sodium chloride, is injected intravenously there is a fall of intralabyrinthine pressure following the fall of cerebrospinal fluid pressure, again showing lag in reaching the final low level. There is a marked loss of intensity for all tones. These experiments prove the intimate relation between intralabyrinthine and cerebrospinal fluid pressures and any changes are in the same direction. In these animal experiments, of course, tinnitus could not be observed.

The intravenous use of hypertonic solutions to bring about a fall in cerebrospinal fluid pressure has become a well established neurosurgical practice since the experiments of Weed and McKibben (1919). Glucose is considered to be one of the most satisfactory solutions for clinical use. The whole question of intracranial circulation and pressure is receiving widespread attention at the present time with the result that there are many articles in current physiological and neurological journals. It is for the most part agreed that in the majority of cases 50 per cent glucose intravenously does cause a fall in intracranial pressure (Browder 1930). It has recently been shown that the initial fall in pressure after glucose injection is followed by a late rise (see Bullock, Gregersen and Kinney 1935), but this does not affect the experiments which we are describing as it occurs several hours after the injection.

The intravenous injection of glucose might be expected to affect tinnitus for at least two different reasons; either because

of the fall in intracranial pressure and its possible effect upon the intracranial circulation; or secondly because of the fall in intralabyrinthine pressure which should occur according to the experiments of Hughson.

An intravenous injection of 100 cc. of 50 per cent glucose was given. The blood pressure was taken before the experiment, immediately after the injection and then at intervals for an hour (see Table 5). Spinal puncture was not done as the patients were not admitted to hospital. The patients were kept lying down. During the hour after the injection, the patient was repeatedly asked about the tinnitus and was instructed to report any change which might take place during the next twenty-four hours.

The injection was carried out in ten cases. In seven there was no change in the tinnitus, in one case the tinnitus was less, but repetition of the injection made the tinnitus worse. Two cases felt that the tinnitus was improved, but in no case was the improvement at all striking, the noise never entirely disappeared.

One must conclude that in these cases lowering of intracranial or of intralabyrinthine pressure had very little effect upon the tinnitus. The one case of Ménière's syndrome in the series was not available for the glucose injection.

TABLE 5 — *The Record of the Blood Pressure Following the Intravenous Injection of Fifty Per Cent Glucose Solution*

CASE NO.	B.P. BEFORE INJECTION S.D.	B.P. IMMEDIATELY AFTER INJECTION S.D.	B.P. ONE HOUR AFTER INJECTION S.D.
1	115/76	120/80	95/65
3	134/84	156/86	118/70
4	120/80	120/82	
5	118/80	122/80	110/74
7	120/90	130/90	128/90
8	158/88		158/86
9	136/84	124/78	114/76
10	120/76	126/82	120/78
16	132/78	140/84	142/88
18	128/84	128/70	114/70

Constricting Neckband — Fowler (1913), discussing the determining factors of tinnitus suggests that some cases of tinnitus are improved by applying a constricting band to the neck. In

our patients the constricting band of a blood pressure apparatus was wrapped around the neck and a pressure of 30 mm. of mercury was maintained for three minutes. This was tried in twelve cases and there was no change in nine; three were made very slightly worse. Fowler states that when tinnitus is increased, the determining factors are in the middle ear and are more or less influenced by treatment. Two of our cases that were made worse were diagnosed as traumatic nerve deafness and the third as middle ear and nerve deafness.

VASOMOTOR CONTROL OF THE CEREBRAL CIRCULATION

White (1935) points out in his monograph on the autonomic nervous system that H. Forbes and S. Cobb and their co-workers have shown active vasomotor control of the cerebral vessels (1928-29). "They observed up to a 50 per cent increase in the capillary bed after sympathectomy and a corresponding decrease on stimulation of the cervical sympathetic nerves." These are the vasoconstrictor fibres to the brain.

Cobb, Finesinger, Chorobski and Penfield (1932) have shown that vasodilator fibres pass up the vagus nerve to the medulla and proceed to both facial nerves as far as the geniculate ganglion where they leave the facial and pass through the great superficial petrosal nerve to the internal carotid artery where they are then carried to the cerebral vessels, just as the sympathetic nerves are. They state that this is a pathway of efferent impulses and that electrical stimulation at any point along the tract produces dilatation of the arteries of the pia mater. This innervation is antagonistic to the cervical sympathetic and may be termed parasympathetic. It is apparent, therefore, that the cerebral vessels are under the control of the sympathetic and parasympathetic nervous systems. Constriction of the vessels and decrease of circulation is caused by stimulation of the sympathetic nerves. Stimulation of the parasympathetic nerves causes a dilatation of the cerebral vessels and increase of circulation as observed by the above group.

In discussing the essential differences between the parts of the autonomic nervous system, White refers to a helpful description of Cannon's: "The functions of the sympathetic nerves are catabolic and give rise to an extraordinary liberation of body energy, they are of a spend-thrift character. Of equal importance to the body are the anabolic functions of the parasympathetic

nervous system which come into play during periods of rest and recuperation and are of a conservative character." This description makes it easier to understand the general effects which follow stimulation or depression of these systems.

DRUGS WHICH AFFECT THE VASOMOTOR MECHANISM

Since it is possible to influence the vasomotor mechanism by the use of drugs which act specifically upon the sympathetic and parasympathetic nervous systems, it should be possible similarly to influence the cerebral vasomotor mechanism and circulation.

We selected the following drugs for our experiments:

Sympathetic	*Stimulant* — Ephedrine hydrochloride *Depressant* — Ergotamine (Gynergen, Sandoz)
Parasympathetic	*Stimulant* — Mecholyl (Acetyl-beta methylcholine Chloride, Merck) *Depressant* — Bellafoline (Sandoz)

White (1935) has observed that the clinical use of these drugs is very limited and the duration of activity very short. "Nearly all act with great intensity on the entire craniosacral or thoracolumbar divisions. As the price of securing one desired result, many disagreeable or even dangerous effects are unavoidably produced." Naturally, one must obtain the full physiological effect before any conclusions can be drawn as to any particular action of the drug. In fact, we used the general reaction — pulse change, etc., of each drug as the indication of the necessary dosage and it was the height of the effect that we considered to be the most important time for observation of the effect upon the head-noises. Furthermore, it should be pointed out that sometimes the general effect of the drugs is so severe and disturbing to the patient that it becomes difficult for him to concentrate upon the tinnitus.

Stimulation of the Sympathetic Nervous System by Ephedrine Hydrochloride — The general effects of the drug in mammals as described by Chen and Schmidt (1930) are as follows: ephedrine in suitable doses raises the blood pressure, constricts the blood vessels, increases cardiac activity, dilates the pupil, relieves broncho-spasm, contracts the uterus and more frequently inhibits than stimulates the gastro-intestinal tract.

By giving ephedrine we expected to produce a vasoconstriction of cerebral vessels and a resulting decrease in the cerebral circulation as noted by Forbes and Cobb after direct stimulation of the sympathetic nerves.

We first used ephedrine hydrochloride, ½ grain, by mouth, giving about 1½ grains a day over a period of two weeks. Because of the uncomfortable general reaction, some of the patients were most irregular in taking the medicine. They were instructed to keep a record of the effect upon the head-noises. In only five cases was the medicine taken with sufficient regularity to record definitely. The remainder of the patients found the general symptoms too distressing to persist. Of the five patients, one was made worse and in four the noise was less. We then changed to ephedrine hydrochloride hypodermically, 1 cc. containing .05 gramme, and the patients were observed for an hour following the injection. The chief physiological change noted after the injection was an appreciable increase in pulse rate, accompanied by flushing and nervousness (one patient became hysterical). These signs were taken as the indication of a full physiological effect. There were seventeen cases injected and in many the injection was repeated on a second occasion if there was any doubt about the first result. In nine cases there was no change in the tinnitus; in two the noise was made worse; in six cases there was some improvement. Three of the patients who were improved when the drug was given by mouth were also improved by the hypodermic injection. In Case 12, there was a marked improvement in the noise over a period of days. The hypodermic injection has been repeated three times and each with improvement. Medication by mouth in this case also improved the noise, but the improvement has not been sustained. However, there is a mental attitude in this patient which makes the result questionable.

In considering the beneficial effects which have followed the administration of ephedrine, which presumably causes pial vasoconstriction, it is possible that we are actually getting the reverse effect, a vasodilatation of the pial vessels accompanied by a rise in blood pressure. It has been shown (Forbes and Wolff 1928, and Forbes, Finley and Nason 1933) that whereas epinephrine applied locally causes a vasoconstriction of the pial vessels, when it is injected intravenously or into the carotid in animals it causes a vasodilatation of the pial vessels accompanied by a rise in systemic blood pressure. This may be the

case (but not necessarily so) with ephedrine, since Chen and Schmidt (1930) have shown that a rise in blood pressure does follow its injection. It has been suggested to one of us by Dr. W. V. Cone that improvement in the tinnitus following the injection of ephedrine may be in some way connected with the blood pressure rise.

Depression of the Sympathetic Nervous System by Ergotamine Tartrate—Rothlin (1929) concludes that ergotamine, which is chemically and therapeutically superior to ergotoxine, paralyzes the augmentor and inhibitory functions of adrenalin on the sympathetic.

Lennox, Gibbs and Gibbs (1935), by means of a thermo-electric flow recorder introduced into a neck vein in a series of patients, observed the effect of intravenous injection of ergotamine tartrate (Gynergen, Sandoz) upon the cerebral blood flow. They found a moderate increase in cerebral blood flow secondary to an increased systemic blood pressure. There was a moderate increase in cerebrospinal fluid pressure. Pool and Nason (1935) studied the effect of intravenous injection of ergotamine tartrate upon the pial vessels in anesthetized cats. They found that the effect was very inconstant upon the calibre of the pial arteries, but there was a constriction of the dural arteries and skin arteries. There was an elevation of systemic blood pressure, an elevation or no change in cerebrospinal fluid pressure. With reference to the pial arteries: in one-third of the cases there was vasodilatation (which should be expected from depression of the sympathetic nerves); in one-third vasoconstriction, and in one-third no effect upon the pial vessels. However, the effect which Lennox and his co-workers obtained in their patients was a moderate increase in cerebral blood flow.

Ergotamine tartrate (Gynergen, Sandoz) was at first given by mouth in doses of 1 milligram tablets, amounting to a dosage of 2 to 3 milligrams per day, for a period of one to two weeks. Twelve patients were given this medication. There was no change noted in the head-noise in five cases; five were made worse and two were improved.

Ergotamine was then given hypodermically in sterile ampoules of 1 cc. containing .5 milligram. This dosage was found to produce severe illness characterized by slow pulse, headache and abdominal colic and vomiting. For this reason, the dosage was reduced to ½ cc. or .25 milligram. The patients were

observed for an hour following the injection. In all cases, there was an appreciable decrease in pulse rate and this was considered as the indication of physiological action. The preparation was given to seventeen patients. There was no change in eleven. The noise was more marked in four and was less in two. Expecting ergotamine to increase cerebral circulation, we had hoped to obtain a more definite effect upon the tinnitus.

Stimulation of the Parasympathetic Nervous System by Mecholyl — The general physiological properties of this drug, as described by Starr, Elsom and Reisinger 1933, Abbott 1933, and by Starr 1933, are as follows: mecholyl (Acetyl-Beta-Methylcholine Chloride, Merck) slows the heart, lowers blood pressure, contracts the bronchioles, increases glandular activity and intestinal tone, stimulates peristalsis and dilates the peripheral vascular system.

We have not been able to find a report of any work which records the effect of mecholyl on the pial vessels. Wolff (1929) has observed the effect of acetylcholine of which mecholyl is a derivative. He finds that the intravenous injection of acetylcholine causes a dilatation of the cerebral arteries, veins and minute vessels, usually a rise in cerebrospinal fluid pressure and a fall in systemic arterial pressure. This is merely an indication of what may follow the subcutaneous injection of mecholyl, but it is not necessarily the case.

We used the drug by mouth in two cases only, 200 milligrams being given every four hours for four days. There was no evidence of parasympathetic stimulation, nor was there any effect upon the head-noise. It was decided that hypodermic administration of the drug would be more satisfactory as we could observe the patient while the drug was effective. Following subcutaneous injection, its effect is supposed to last fifteen to twenty minutes. We found that in most cases a dosage of 5 milligrams produced a definite physiological reaction in about three minutes; marked flushing, salivation, slight fall in blood pressure and an appreciable rise in pulse rate occurred. In some cases, a dosage as high as 15 milligrams was given, but we found that the most satisfactory dosage is 5 milligrams, because the general reaction is not so extreme. The drug was repeated when thought necessary and the patient observed for an hour. It was given to eighteen patients, twelve showed no change, three were made worse and three were improved. All of the cases

showed undoubted generalized effect of parasympathetic stimulation.

Expecting this drug to stimulate the parasympathetic nerves and thus increase cerebral circulation, it was disappointing not to find a more definite effect upon the tinnitus.

Depression of the Parasympathetic Nervous System by Bellafoline — Bellafoline (Sandoz) is the total levorotatory alkaloidal complex of bellafoline. Cushny (1904) and Rothlin (1924) have studied the effect of atropine and bellafoline upon the autonomic nervous system. White (1935) outlines the pharmacological action of atropine as follows: atropine paralyzes the parasympathetic nerves. It inhibits the secretion of salivary, gastric, pancreatic and mucous glands, dilates the pupil and prevents slowing of the heart when the vagus is stimulated. The sweat glands are also paralyzed. We have been unable to find a description of the direct action of atropine or bellafoline upon the cerebral vessels or cerebral circulation.

Bellafoline tablets (.25 milligram) were given by mouth up to 1 or 2 milligrams a day for a period of one or two weeks. This procedure was carried out in thirteen patients. There was no effect upon the tinnitus in four; one was made worse and eight were improved. The drug was then given hypodermically, — an ampoule containing 1 cc. (.5 milligram). The patients were observed for an hour. The full physiological effect was determined by the occurrence of dryness of the mouth and an appreciable increase in the pulse rate. The drug was repeated when necessary until the definite physiological effect was obtained. There were seventeen patients so treated. There was no change in the head-noise in ten of the cases; two were made worse, both of which had been improved by the mouth administration. Five were improved. Of the five improved, one had been made worse by the mouth administration and one had not been affected. It is interesting to note that of all the drugs used, bellafoline by mouth caused a lessening of the tinnitus in the highest number, but this was not confirmed by carefully controlled hypodermic injection, which though of shorter duration, at least produces the full physiological effect of depressing the parasympathetic nervous system.

By paralyzing the parasympathetic nerves, this drug should eliminate the cerebral vasodilator effect which Chorobski and Penfield have demonstrated, thus tending towards vasoconstriction and diminished cerebral circulation. It is worthy of note

that the two drugs, ephedrine and bellafoline, which might be expected to induce cerebral vasoconstriction, have had a more beneficial effect upon the tinnitus than have the drugs which induce vasodilatation.

Simultaneous Depression of the Sympathetic and Stimulation of the Parasympathetic Nervous Systems — The object of this was to increase the vasodilator effect of the parasympathetic system by paralyzing the sympathetic nerves at the same time, in the hope of getting a maximum vasodilatation and increased cerebral circulation. For this purpose, ergotamine ½ cc. (.25 milligram) was injected subcutaneously, and as soon as there was an appreciable decrease in the pulse rate, 5 milligrams of mecholyl was injected subcutaneously. This procedure was carried out in twelve cases. The final result was that in eight cases there was no change in the noise. In three cases, there was improvement of the head-noise and in one case the noise was worse. This result on the whole was disappointing as we hoped that an increase in cerebral vasodilatation would cause a lessening of the tinnitus.

Simultaneous Depression of the Parasympathetic and Stimulation of the Sympathetic Nervous Systems — This should bring about a maximum vasoconstrictor effect upon the cerebral vessels with a resulting diminution in cerebral circulation.

The procedure was, first, to inject bellafoline and follow it with ephedrine. Bellafoline 1 cc. (.5 milligram) was injected and as soon as there was dryness of the mouth and an increase in pulse rate, ephedrine hydrochloride 1 cc. (.05 gramme) was injected subcutaneously. This was carried out in eleven patients. The final results were that four cases showed no change in the noise. In two cases, the noise was worse. Five cases were improved, some of them being much improved.

Five of these cases were improved as compared with only three from the ergotamine-mecholyl combination of drugs. As was the case when the drugs were given separately, it is the combination which tends towards cerebral vasoconstriction which has had the most beneficial effect upon the tinnitus.

CERVICAL SYMPATHECTOMY

Brunetti (1933) in discussing the effect of cervical sympathetic operations on certain ear conditions refers to what he considers to be the first sympathetic operation for an ear con-

dition. Ferreri in 1903 did an extirpation of both superior cervical ganglia in a man who suffered for a long time with most troublesome tinnitus. The result was satisfactory and lasting. He mentions another case, one of Calgero's, in which the cervical sympathetic ganglion on one side was removed in a young girl who suffered from head-noises and a severe bilateral deafness. There was a decrease in the noise, but no improvement in the hearing. Brunetti states that in his own experience he could not think of any case in which he had obtained extraordinary benefit as far as head-noises are concerned.

Removal of the Stellate Ganglion — Since vasomotor control of the cerebral vessels has been demonstrated, the effect of complete removal of the cervical sympathetic nerves on one or both sides must be considered. It was felt, therefore, that a study of cases upon which stellate ganglion removal had been done might show some alteration in head-noises, if present before the operation. One of us, L. H. M., had performed the operation of stellate ganglion removal for various conditions in twelve cases, some of which had tinnitus previous to the operation. Of the twelve cases operated upon, reports were obtained from eight. Of these eight, four admitted having head-noises before the operation; and of these four, two stated that they no longer had head-noises. The third was decidedly improved, and the fourth stated that the noises were neither better nor worse. Of course, this is a very short series, yet it does furnish evidence that the operation of stellate ganglionectomy has improved tinnitus in three out of four cases. With a larger series, one would be able to estimate more definitely whether the operation is justified in cases of tinnitus.

It should be pointed out that stellate ganglionectomy is equivalent to the sympathectomy of Forbes and Cobb to which we have previously referred, and following which they noted "a 50 per cent increase in the cerebral capillary bed" — a vasodilatation. The improvement in the tinnitus following stellate ganglionectomy is a direct contradiction of the results we have obtained from drug stimulation of the autonomic nervous system.

TINNITUS INCREASED

The procedures which most often caused an increase in the tinnitus were deep breathing, inhalation of amyl nitrite and the injection of ergotamine. These are not all considered to have the same effect upon the cerebral blood flow.

SUMMARY

Tinnitus may be caused by a lesion in the nerve tissue in Ménière's syndrome, in intracranial disease and possibly in otosclerosis. However, in some proven cases of cochlear disease, Crowe and his co-workers did not find tinnitus to be a prominent symptom, though it was occasionally present.

In a series of 351 cases of intracranial disease, tinnitus was present in 10 per cent of the cases in which there was no obvious local ear disease.

Experiments were carried out in a series of 19 patients suffering from tinnitus; the ages of the patients vary from 19 to 52 years; the duration of the tinnitus varies from a few months to about 30 years; and the diagnosis of the ear lesions covers a wide range. However, an examination of Table 2 shows that some of the youngest patients, e.g. (Case 1), or ones who have had the tinnitus for a very short time, e.g. (Case 10), did not show as much improvement from the various procedures as did older patients, e.g. (Case 15), or the ones who have had the tinnitus longer, e.g. (Case 8).

Both ephedrine and bellafoline caused improvement in the tinnitus in a greater percentage of cases when given by mouth than when given hypodermically. Since the hypodermic method of administration was more accurately controlled, its results are probably more reliable.

Stimulation of the sympathetic or depression of the parasympathetic nervous system by drugs whether carried out separately or simultaneously had a slightly more beneficial effect upon the tinnitus than did depression of the sympathetic or stimulation of the parasympathetic nervous system. Yet the operation of stellate ganglionectomy (depression or elimination of the sympathetic) caused an improvement in the tinnitus in three out of four cases.

In the present series of nineteen cases, all the procedures carried out with the exception of the sodium-free diet were directed towards alteration of the cerebral circulation or cerebral pressure. We have no direct proof of the nature of the alteration produced since we made no records of cerebral circulation or pressure during the course of the experiments. It is most likely that the cerebral circulation was affected because the drugs used produced their full physiological effect, and most of these drugs have been shown by other experimenters to have a definite effect upon the cerebral vascular mechanism.

It is important to note that, in spite of an undoubted alteration of cerebral circulation, the tinnitus in the majority of the cases in this series was unaffected by the procedures employed.

Detailed conclusions in this preliminary report are not justified, but, as the experiments proceed, efforts will be made to lengthen the series, to eliminate some of the variables and to control methods of procedure as far as possible.

* * *

Our grateful thanks are due to the American Academy of Ophthalmology and Otolaryngology, which by providing W. J. McNally with an annual research fellowship during a period of years has greatly contributed to the promotion of the work here reported. For advice as to the effects of certain drugs upon the cerebral circulation, we wish also to express our indebtedness to Professor R. L. Stehle, of the Department of Pharmacology, and to Dr. Nathan Norcross, of the Department of Neurosurgery, McGill University. For help in searching case reports, we are greatly indebted to Dr. Gilbert Holland, Royal Victoria Hospital. For kindly supplying us with drugs we are greatly indebted to the following companies: The Abbott Company, Limited, for ephedrine hydrochloride; The Merck's Chemical Company for mecholyl, and The Wingate Chemical Company for ergotamine and bellafoline.

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DISCUSSION

DR. L. K. GUGGENHEIM, St. Louis: After hearing this splendid dissertation upon the subject of tinnitus, one is impressed with the fact that the ear consists of more than the temporal bone. It seems rather trite to say that, but for thirty years I have been studying the temporal bone and have been visiting laboratories such as the splendidly equipped one at Johns Hopkins, and wherever one goes one is shown beautiful specimens of abducens, and when one asks about the eighth nerve, the vestibularis nuclei in the brain stem, the anterior cochlear nucleus, and so on, arms are thrown up and the answer is, "Well, we just haven't gotten to that."

I want to make a plea, or to stimulate a plea, that we become more interested in the anatomy of the intracranial pathways and that our laboratories start to investigate the autopsy material for conditions other than the intraparietal organ.

Dr. McNally brought out the work of Gray, who has found no changes in the organ of Corti, but has found marked changes in the membranous cochlea. That was my chief reason for getting up. I felt that I should announce the fact that in our Institute in St. Louis we have very carefully examined the membranous cochlea in all our cases of otosclerosis, and we have not been able to find any evidence of the degeneration on which Dr. Gray reported. Our series, of course, is small. It is a tremendously important thing to have this report of Gray's substantiated. I would be very happy to hear, at a later date, from anyone who has found evidence of these changes in the membranous cochlea. We have found absolutely none.

FOCAL INFECTION IN ARTHRITIS*

D. M. Lierle, M.D.

Iowa City, Ia.

This paper is a preliminary report of a statistical study that is being carried on jointly by the various departments concerned with the problem at the University Hospital. An attempt is being made to evaluate the therapeutic effect secured by removal of foci of infection in arthritis. This particular report concerns the study of 148 cases of chronic, non-specific arthritis in adults, and includes findings from a previous study of 37 children under 16 years of age.

In the selection of adult cases, the following points were considered: (1) presence of well-established joint lesions; (2) assurance that the disease had been present for at least two years. (The study was limited to those cases in which careful post-operative observations could be made over a period of from two to four years following treatment.) (3) Limitation of cases, as far as possible, to those in which arthritis was the only systemic affection. This rather small group was selected from a larger one of over 800 cases.

Billings[1] defines a focus of infection as a "circumscribed area of tissue infected with pathogenic micro-organisms." He further states that the foci may be primary and secondary. Primary foci are usually located in tissue communicating with a mucous or cutaneous surface. Secondary foci are the direct result of infection from other foci through contiguous tissues or at a distance through the blood stream or lymph channels.

That systemic or general disease may be due to a focus of infection has long been an established conclusion. Benjamin Rush,[2] as early as 1801, reported a case of rheumatism which disappeared following the removal of an infected tooth. Twenty years ago, Billings,[1] Rosenow[3] and others directed our attention to the problems of focal infection. Today, the principle of focal

* From the Department of Otolaryngology and Oral Surgery, University Hospital, Iowa City, Iowa.

infection is more or less accepted. However, the fact that a bacteriemia exists and is maintained by a focus of infection is not so generally accepted. Some observers have reported positive blood cultures in as few as three per cent of cases of arthritis; while others have reported as high as 60 per cent.

In this particular study, the cases were divided into two main types: Atrophic or chronic infectious arthritis, and hypertrophic or osteo arthritis, according to the classification adopted by the American Committee for the Control of Rheumatism.

Cecil[4] describes atrophic arthritis as a "chronic disease of the joints, characterized by inflammatory changes in the synovial membrane and periarticular structures, and by atrophy and rarefaction of the bones. In the early stages, the disease manifests itself as a migratory swelling and stiffness of the joints; in the later stages by more or less deformity and ankylosis. Rarely one joint is affected; in the majority of cases the disease is polyarticular."

The first group with which this study is concerned consists of 126 cases of atrophic arthritis, 37 children, and 89 adults.

The younger group of 37 has been reported upon previously by Miltner and Kulowski[5] of the Department of Orthopedics. The disease was polyarticular as a rule and usually involved one or more of the larger joints. Ten in this group had advanced Still's disease, with enlargement of the liver, spleen and lymph nodes.

Foci of infection in this group were found exclusively in the upper respiratory tract. Seventy-three per cent (27 cases) were apparently free from symptoms following the attempt to eradicate foci of infection; 16 per cent (6 cases) were markedly improved; 11 per cent (4 cases) showed no improvement. The last-named were all cases of Still's disease.

	Per cent	*Cases*
Free from symptoms	73	27
Marked improvement	16	6
No improvement	11	4

Chronic faucial and pharyngeal tonsillitis were present in 24 cases. Paranasal sinus disease and chronic otorrhea were present in 6. No dental foci were found.

The adult cases in the atrophic group revealed 6.8 per cent (6 cases) free from symptoms following treatment; 42.6 per cent (38 cases) markedly improved; 25.8 per cent (23 cases) moderately improved and 24.7 per cent (22 cases) not improved.

	Per cent	*Cases*
Free from symptoms	6.8	6
Marked improvement	42.6	38
Moderate improvement	25.8	23
No improvement	24.7	22

Positive histories of upper respiratory infection were given in 75 cases; 14 gave negative histories.

This so-called silent group, comprising those cases which did not have histories of coincidental upper respiratory infection gave results comparable to those of the general group.

Cecil[4] describes osteo arthritis or hypertrophic arthritis as a "chronic arthropathy, occurring usually in elderly people and characterized by certain degenerative and hypertrophic changes in the bone and cartilage. In some cases there is considerable thickening of the synovial membrane."

The results following attempts to eradicate foci of infection in the hypertrophic group were unsatisfactory, merely substantiating the conclusion that any improvement was due to the improvement of the general condition. There were, only in the adult group, 59 cases of hypertrophic arthritis. None were completely relieved of symptoms. Marked improvement was noted in 12 cases, or 20.3 per cent; 8 cases, or 13.5 per cent of those studied, were moderately improved; 39 persons, or 66.1 per cent showed no improvement.

	Per cent	*Cases*
Relieved of symptoms	0	0
Marked improvement	20.3	12
Moderate improvement	13.5	8
No improvement	66.1	39

In the hypertrophic group, 49 cases had positive histories of upper respiratory infection and 10 gave negative histories. The results of the silent group were about the same as those of the larger group.

The following operative procedures were done on the 148 adult patients:

Tonsillectomy	136
Submucous resection	78
Adenoidectomy	5
Caldwell-Luc operation	29
Spheno-ethmoid evisceration	10
Antrameatal	4
Lingual tonsil cautery	15
Extraction of teeth	30

Discussion

In the present study, the best results were obtained in the group of children. The explanation of this may be that cases were not as long-standing as the adults', and that in many instances a single focus of infection was present and easier to eradicate. Only five children had advanced Still's disease, and four members of this group were not benefited.

The results in the adult study were somewhat disappointing, particularly in the hypertrophic group. It is more or less generally accepted that focal infection plays little or no part, as an etiological factor in hypertrophic arthritis. Any improvement, in our opinion, may be accounted for on the basis of the improved general condition which might follow treatment of infection in the upper respiratory tract of any patient.

The response in the atrophic group was somewhat better. The generally poor results obtained may be due to the fact that the majority of cases in this group were of long standing and multiple lesions were present. Most of the cases had multiple foci which in many cases were not easily eradicated. There are a number of observers who believe that in many instances the joint itself acts as a focus. It is quite evident that if this is true, very little improvement might result in some cases following the treatment of foci.

Steindler,[6] Kulowski and Miltner[5] believe that unsatisfactory results in atrophic arthritis may be due to the fact that the joint, especially the synovial membrane, may be considered as a secondary focus of infection.

Kulowski and Miltner[5] report 70 synovectomies performed on arthritis of the atrophic type in which the pathological struc-

tures indicated the chronic inflammatory nature of the reactions in the excised tissues. It was evident that in general the best results were obtained in those cases which were treated in the early course of the disease.

It is our observation that infected tonsils and teeth appear to be responsible foci more often than chronic paranasal sinus disease. However, sinus infection in some instances certainly provides a focus. Some observers have emphasized the importance of mild hyperplastic paranasal sinus disease. Our earlier observations failed to justify radical procedure except in the more advanced cases of paranasal sinus disease.

It is surprising to note the lack of direct relationship between acute upper respiratory infections and the onset of arthritis. In this particular study such a positive history was obtained in only 3 per cent of the cases.

The treatment of arthritis certainly concerns nearly every field of medicine. The cooperation and intimate association of the departments concerned are very necessary. The otolaryngologist certainly should have definite information regarding the type of arthritis, the extent of the joint pathology and a thorough knowledge of the patient's general condition.

The patients in the group observed seem to show better results if the operative procedures were done during periods of quiescence, and when several operations were necessary the best results were obtained if sufficient time elapsed between procedures.

In conclusion, it appears that (1) arthritis in children responds very well to the eradication of focal infection in the upper respiratory tract; (2) atrophic arthritis in adults does not show the marked response seen in children, but in selected cases the treatment is fairly satisfactory; (3) hypertrophic arthritis shows only questionable response to removal of foci which well may be solely the result of the general improvement following the treatment of the upper respiratory infection.

A much larger series of cases, observed over a longer period of time, is necessary before definite conclusions can be made.

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DISCUSSION

DR. JOHN J. SHEA, Memphis, Tenn.: Dr. Lierle has given a concise opinion upon the findings of focal infection in arthritis as studied at the University Hospital of Iowa. I agree with him in all of his conclusions. As one of the consultants of the Willis C. Campbell Clinic for Orthopedics, it has been our experience, that in the children we get our best results, provided that we exclude the cases of Still's disease. The atrophic arthritis adult patient is always helped in a general way by the removal of any or all of the focal infections, provided that the established joint pathology is not so advanced as to serve as a secondary focal infection. We find that the patients with true hypertrophic arthritis are not materially benefited in their joints by the removal of any existing focal infection.

The selection of the patient to be submitted for operation must depend upon the joint opinion of the orthopedic surgeon, the internist and the otolaryngologist. After the surgery has been completed, these patients should remain under the care of the orthopedic surgeon and the internist and be content to carry on with a partial disability.

DR. HENRY M. GOODYEAR, Cincinnati: In selecting 148 cases from a total of 800, Dr. Lierle has been exceedingly careful to report only cases of true arthritis. A number of the cases selected had previously received intensive internal and physical therapy; thus, those recovering after the removal of foci helped to emphasize the importance of this procedure.

The statement is not without justification "know arthritis and one knows medicine," so extensive are the accompanying bodily changes.

In session, physicians should use a common parlance. We should recognize at least two great types, namely, atrophic and hypertrophic arthritis. It is important to distinguish between these two forms as their etiology is probably quite different; the symptoms are not the same; their response to the removal of foci is not identical, and they are quite different as to prognosis.

The terms atrophic and hypertrophic are based on x-ray findings and from a clinical standpoint are rather misleading. To an obese patient past forty years of age with arthritis of the knee (a weight-bearing joint) we would expect to apply the term atrophic arthritis or pressure atrophy. However, roentgenographs would show a proliferation of bone and the case would be classed as a case of hypertrophic arthritis.

I wish especially to emphasize that atrophic arthritis occurs most commonly in young people. The lesion is progressive and the clinical picture is one of multiple painful swollen joints, often red and acutely inflamed. It is this lesion that is most apt to respond to the removal of foci of infection, teeth, tonsils and sinuses, in the order named, so far as my observation has been able to discern.

Patients responding well to the removal of tonsils more frequently gave the history of an acute upper respiratory infection and sore throat within two or three weeks prior to the onset of multiple joint pains.

Not infrequently, upon examining a patient with atrophic arthritis, you find an upper molar or a pre-molar tooth is absent. Upon questioning, the recovery following extraction was slow and painful accompanied by some nasal disturbance which had recurred with each succeeding attack of acute rhinitis. In the year following the extraction, joint pains appeared and were progressive in character. A study of the nasal sinuses reveals a chronically infected antrum, the correcting of which offers considerable hope to the arthritic.

I have rarely seen any improvement following the removal of suspected foci in cases of hypertrophic arthritis. The patient is usually fat and past forty years of age. The weight-bearing joints (knees, ankles, and metatarsal) are the chief sources of trouble. Often only one knee is affected which may be painful but not acutely inflamed. The disease is not progressive and a metabolic disturbance probably plays a greater rôle than foci of infections.

Multiple joint infections in children often require repeated and determined effort to free the throat by knife or cautery of all lymphoid tissue in the throat and nasopharynx. Large intranasal antrum openings and even exenterations of the ethmoid cells and opening of the sphenoids may be justified.

THE RELATION OF DIET TO CERTAIN OTOLARYNGOLOGIC PROBLEMS

CECIL STRIKER, M.D.
BY INVITATION

CINCINNATI

I SHOULD LIKE to take this opportunity to thank the officers of this Society for extending to me an invitation to appear before you. My purpose in this paper is to discuss certain metabolic principles in relation to otolaryngological pathology.

Bacteriological and morphological factors in disease have been stressed for years in all branches of medicine,—this almost to the exclusion of other factors—but now we have equally if not more important, approaches to disease. A study of the physico-chemical or metabolic changes yields interesting and useful knowledge in the understanding and treatment of diseases. That diet and local environment play a rôle in disease is evidenced by the occurrence of urinary calculi. In Great Britain, "stone areas" are well recognized; urinary calculi occur more frequently along the east coast, and in parts of Derbyshire and in North Wales. Joly stated that in the east and southeast of France, kidney and bladder stones are prevalent, but are found infrequently in Normandy. In Africa, urinary calculi frequently occur in the region of the lower but not the upper part of Egypt. McCarrison observed the frequency of stones in regions in which there was not proper balance of food constituents in the diet. Therefore, as it is in urology, so probably it is in otolaryngology.

Our food now is preserved, purified, polished, pickled, canned, extracted, distilled, concentrated, heated, dried, frozen, thawed and stored. How are we to select the food that is good for us?

Through painstaking observations and experimentation we have found that man's diet can be analyzed into the following components: Carbohydrates or sugars, for food energy; fats for heat; proteins for body building; and water, mineral salts and vitamins with their multitude of functions.

Just as in the specialty of otolaryngology there is specialism, so also in the field of diet therapy there is specialism. Today we have the gastroenterologist, the allergist, the infant feeder, the vitamin expert, etc. Both time and ability prevent my discussing all phases, therefore, I wish to confine my remarks to certain metabolic principles and their relation to otolaryngology.

We are all aware of the wide range between man's need for food and his actual intake. Body requirement of caloric intake is based on age, occupation, sex and square meters of body surface. It has been found that under basal conditions the body requires 25-30 calories per kg. of body weight and this may rise to 80 cal. per kg. of body weight, depending on the type of work. In addition to the caloric intake one must have a knowledge of the type of food that supplies the calories. There is an irreducible minimum of protein required. This is due to the fact that, unlike fat and carbohydrate, protein is not stored —a minimum of one gram of protein per kg. of body weight is advisable.

There must be a definite relation between the amount of carbohydrate and fat combusted. Incomplete combustion of fats produces ketone bodies which may lead to an organic acid acidosis. In addition to the above there are basal requirements for certain minerals, such as Fe, I, Mg, Ca, P, Na, Cl, etc.

From the above, one can see that many known and unknown chemical reactions take place. A proper chemical balance makes for good health, whereas disease arises when there is an alteration. I shall not attempt to go into all of these complex chemical changes, but will discuss acidosis, alkalosis and some phases of mineral metabolism.

Very considerable amounts of acids are constantly being produced in the body during the process of normal metabolism. These acids, like all others, owe their activity to the hydrogen ions liberated in solution. These hydrogen ions are capable of bringing about marked physiologic changes or even death. Fortunately, the body possesses a very effective mechanism for rendering the hydrogen ions ineffective. The degree of acidity or alkalinity within the various cells of the body differs slightly from that of the blood, but the blood reflects the reaction throughout the body.

Protoplasm has a very high buffering power — and if its p_H value changes significantly the cell rapidly dies. At the same

time it is not easy to harmonize these conclusions with evidence from other sources. An acid such as CO_2 undoubtedly enters the living cell — so that if the hydrogen ions accumulate in discreet granules, it is these granules which must be responsible for the apparent buffering power of cytoplasm. However efficient the granules may be as absorbents of hydrogen ions, there must come a time when no more acid can be absorbed — and at all times there must be an equilibrium between the amount of acid in the granules and the amount in the cytoplasm. Consequently, there must, theoretically, be a change in the cytoplasm whenever the total amount of acid in the cell is varied. This change may not be detectable by indicators, but it is possibly of profound physiological importance. Practically every physiological activity is sensitive to the presence of acids which penetrate the cells. Ciliary and amœboid movement, mitosis, cell division and respiration are profoundly depressed by the presence of intracellular CO_2 or any other rapidly penetrating acid. Therefore, in view of our inability to detect these changes we can only interpret the changes that are measurable by our present chemical knowledge.

Van Slyke has defined acidosis as a condition in which the base in the blood available for combination with bicarbonate is diminished, and the opposite condition, alkalosis, as one in which there is present an abnormal excess of base available for combination with bicarbonate.

The body possesses three important agents for regulation of neutrality: bicarbonate, phosphate, and ammonia.

In the normal individual, breathing normally, carbon dioxide is excreted at the same rate as it is being formed, so that there is a fairly constant balance maintained in the blood between carbonic acid and the proportion of that carbonic acid neutralized to bicarbonate by available base. As long as there is an automatic response by the respiratory mechanism to any increased production of CO_2 by the tissues or to any change of concentration of base in the plasma, so that the ration is maintained at a constant value, the p_H of the blood remains unaltered.

Unless there were means of replenishing the depleted blood bicarbonate or means other than respiration for removal of foreign acids from the body, fatal acidosis would frequently occur. Fortunately, there is a mechanism for the replenishment of the bicarbonate. The kidneys are able to secrete urine which is

much more acid than the blood. This is accomplished by the excretion chiefly of acid phosphate, which is formed from a more alkaline phosphate occurring in the plasma.

In the presence of acidosis, the urine is usually more acid than normal. The urine is normally somewhat acid, having an average p_H of about 6.0, although the reaction shifts from time to time during the day, depending upon the intake of food. The occurrence of an alkaline tide after meals is one illustration of the fact that variation in hydrogen ion concentration is in response to changes elsewhere which necessitate such an adjustment for maintenance of equilibrium.

Mineral acids and salts do have an effect on the acid base balance, but fairly large amounts must be administered. Thus, Peters and Van Slyke have shown that 0.026 gm. of $NaHCO_3$ per kg. of body weight (1.5 gm. for an average individual weighing 60 kg.) is needed to raise the plasma carbon dioxide 1 volume per cent. It takes 45 gms. of $NaHCO_3$ daily to render the urine continuously alkaline.

Another mechanism for the neutralization and elimination of foreign acids consists in the production of ammonia from urea, a neutral substance. This ammonia seems to be formed chiefly in the kidneys and is used to neutralize foreign acids which can be eliminated as neutral salts.

TABLE 1.—*Effect of sodium citrate administration upon the acid-base equilibrium of pre-breakfast blood*

Citrate Administration	Period	Urine p_H	Blood Plasma*	
			$BHCO_3$	p_H
			m. Mol. per L.	
Control	Initial	7.2	25.8	7.45
8 gm. citrate daily	7 days	7.8	25.6	7.40
Control	Initial	7.6	26.0	7.45
4 gm. citrate daily	9 days	8.0	25.8	7.45
30 gm. citrate daily	4 days	8.6	28.2	7.48
30 gm. citrate daily	22 days	8.4	27.9	7.48
30 gm. citrate daily	39 days	8.4	28.2	7.45
Control	10 days	6.8	26.5	7.45

* Recalculated for completely oxygenated blood at p_H drawn.

Note constancy of p_H of blood after large doses of citrate (Bischoff, Sansum, et al.).

TABLE 2—*Effect of ash variation in diet upon the pre-breakfast and acid-base equilibrium of the blood*

Diet	Period	24 Hour Urine		Blood Findings (Plasma)*	
		p_H	Titratable Acidity	p_H	$BHCO_3$
			cc. N/10		m. Mol. per L.
Usual	6 days	5.8 ± 0.15	234 ± 27		
Usual	4th day	6.2	295	7.40	29.0
Usual	5th day	5.8	200	7.41	26.9
Basic	9 days	6.0 ± 0.15	130 ± 31		
Basic	2nd day	6.4	68	7.45	27.2
Basic	4th day	7.2	98	7.41	28.8
Basic	9th day	7.2	113	7.44	29.2
Acid	9 days	5.9 ± 0.12	262 ± 17		
Acid	4th day	5.8	273	7.40	28.5
Acid	9th day	6.4	246	7.44	29.2

* Complete oxygenation.

Note constancy of p_H of blood on acid ash and alkaline ash of diet (Bischoff, Sansum, et al.).

While evidence (Bischoff, 1932) indicated that sustained changes in CO_2 content and plasma p_H of humans, outside the so-called normal limits, could not be produced by ingestion of foodstuffs, the possibility and significance of changes within this limit has not been eliminated. However, in a recent study, Bischoff, Sansum and others found no significant difference in the acid base picture of the blood of a normal individual, drawn before breakfast, whether the mixed diet consumed contained excessively acid or excessively alkaline ash foodstuffs. A diet containing 240, 204 and 93 grams of protein on consecutive days and 200 cc. of normal acid equivalent of ash daily produced a doubtfully significant shift in the blood picture to the acid side. Cullen and Earle have recently studied the normal changes in p_H and CO_2 during the course of the day and found that there is a definite tendency toward increased p_H and CO_2 content following meals. There are marked exceptions and the changes are different in degree and regularity following the evening meal as compared to the noon meal. Thus one can see that no conclusive evidence is available to substantiate the belief that certain diets alter the acid base equilibrium of the blood.

Haldane showed that by taking 15-20 grams of NH_4Cl per day an acidosis could be produced. The daily administration of 45 grams of $NaHCO_3$ was found necessary to change the reaction of the blood to a similar extent in the opposite direction. It would require 18 lbs. of oranges to have an alkaline ash content equivalent to the 40 grams of $NaHCO_3$, and 4.5 lbs. of lean beef or 2 lbs. of oysters to have an acid ash content equivalent to 15 grams of ammonium chloride. The equivalent $NaHCO_3$ as alkaline ash of 1 lb. of bananas is 2 grams. One quart of orange juice contains the alkaline ash equivalent of, roughly, 4 grams of $NaHCO_3$.

In recent years the belief has been voiced that respiratory symptoms and pathologic changes in the respiratory system are brought about by faulty acid base metabolism. This group has expressed the opinion that the color of the mucous membrane of the nasal septum is an index of the acidity or alkalinity of the body — pale septums being present in states of alkalosis and red septums in acidosis. They have been treated accordingly — if there is a pale mucous membrane the patient has been given a diet high in acid ash with dilute nitrohydrochloric acid, and if red they have been given an alkaline ash diet with $NaHCO_3$.

In Jarvis' report there is no clinical or laboratory evidence to prove an abnormal acid base equilibrium.

Bernheimer and Cohn took two groups of patients and determined: (1) Whether an abnormally red or pale color of the nasal septal mucous membrane actually indicates a state of acidosis or of alkalosis. (2) Whether patients suffering from various diseases of the respiratory tract are benefited by placing them on an alkaline or acid ash diet, other factors remaining unchanged. No correlation could be found between the color of the nasal mucous membrane and the clinical symptoms of acidosis in fifty critically ill patients, all of whom showed clinical symptoms of acidosis with hyperpnea, dehydration and acetonuria.

In one hundred normal subjects and in twenty patients with various diseases but with normal hydrogen ion concentration of the blood there were found all three types of septal mucosa — pale, normal pink and scarlet red.

They concluded that diets high in foods producing alkaline or acid metabolic products, together with the adjunct therapy, failed to influence the symptoms or conditions.

TABLE 3

Patient	Clinical History	Color of Nasal Mucosa	pH of Blood Serum
1.	Hyperesthetic rhinitis with usual complaints of sneezing, nasal block and watery discharge; duration two years	Pale, waterlogged	7.44
2.	Hyperesthetic rhinitis; usual complaints as above; duration 17 months	Pale, waterlogged	7.41
3.	Hyperesthetic rhinitis; usual complaints as above; duration three years	Pale, waterlogged	7.41
4.	Hyperesthetic rhinitis; usual complaints as above; duration 18 months	Pale, waterlogged	7.46
5.	Hyperesthetic rhinitis; usual complaints as above; duration two months	Pale, waterlogged	7.44
6.	Hyperesthetic rhinitis; usual complaints as above; duration nine months	Pale, waterlogged	7.45
7.	Hyperesthetic rhinitis; usual complaints as above; duration seven months	Pale, waterlogged	7.45
8.	Acute sinusitis; duration ten days	Pale	7.38
9.	Chronic right otitis media with cholesteatoma; duration seven years	Pale	7.38
10.	Seasonal hay fever with pollen asthma	Pale, waterlogged	7.41
11.	Seasonal hay fever	Pale, waterlogged	7.41
12.	Bronchial asthma; duration four years	Pale	7.40
13.	Polypoidal sinus disease; duration three years	Scarlet red	7.44
14.	Postthyroidectomy; duration two months	Normal	7.37
15.	Acute pharyngitis with cervical adenopathy; duration three days	Scarlet red	7.38
16.	Chronic right otitis media; red septum symptom complex; duration two years	Scarlet red	7.45
17.	Secondary laryngitis; red septum symptom complex; arteriosclerosis and hypertension	Scarlet red	7.48
18.	Chronic right otitis media; red septum symptom complex; duration ten years	Scarlet red	7.48
19.	Frequent attacks of sinusitis; red septum symptom complex; duration two years	Scarlet red	7.45
20.	Subacute ethmoiditis; red septum symptom complex; duration one year	Scarlet red	7.41

Various clinical conditions — pale-red septum. Constant pH of blood (Bernheimer and Cohn).

TABLE 4 — *Alkali reserve of blood in nasal conditions (M. Fineberg)*

Group Classification	No. of Cases	Average % CO_2 for Group	Highest % in Group	Lowest % in Group
Chronic purulent sinusitis; Mucosa red	16	60.4	67	53
Hyperplastic sinusitis (no pus); Mucosa red	11	59.6	67	46
Allergic rhinitis (mucoid); Mucosa pale	6	62.3	69	55
Allergic rhinitis (no secretion); Mucosa pale	6	60.2	66	55
Combined allergy and sinusitis	9	57.5	69	52
Acute rhinitis	5	61.9	67	57

Fineberg studied fifty-three cases of various types of nasal conditions in relation to their carbon dioxide combining power. Realizing that it is difficult to alter the p_H of the blood due to the buffer action of the carbonates, and also it is difficult to interpret minor changes in the almost constant p_H, he resorted to measuring the carbon dioxide combining power of the blood which is an exceeding sensitive indicator. In no case was the alkali reserve outside the limits of normal.

He concludes that while clinically empiric treatment of nasal affections may be based on the color of the nasal mucosa, the benefits are not due to any direct relation on the acid alkali equilibrium of the blood.

In collaboration with Dr. Henry Gruener and Dr. Samuel Seltz, the essayist has observed the color of the nasal mucous membrane of hospital patients who have been on controlled alkaline and acid ash diets, and also who have been given 300 grains of sodium bicarbonate and 150 grains of ammonium chloride daily. The urine p_H varied from 4.5 to 8.6 and the CO_2 combining power of the blood varied from 47 vol. per cent to 86 vol. per cent. Over sixty examinations revealed no correlation between mucous membrane changes and the type of diet or therapy instituted.

TABLE 5 — *Effect of ash variation in the diet upon the pH of the urine and CO_2 combining power of the blood and the color of the nasal mucosa*

Diet	Period	Urine pH (fresh) A.M.	Urine pH (fresh) P.M.	Blood CO_2 Vol. %	Nasal Mucosa
Acid Ash	9 days	4.2 to 5.6	4.5 to 6.7	53 to 60	Pinkish
Alkaline Ash	7 days	7.5 to 8.0	7.3 to 8.0	65 to 83	Pinkish
Acid Ash	11 days	4.5 to 5.5	4.5 to 5.0	55 to 65	Pinkish
Neutral Ash	5 days	6.5 to 7.5	6.5 to 7.5	———	Pinkish
Neutral Ash 300 gr. $NaHCO_3$	2 days	8.0 to 8.5	8.0 to 8.5	86	Pinkish
Neutral Ash 150 gr. NH_4Cl	2 days	4.5 to 5.0	4.5 to 5.0	47	Pinkish

Doctors S. Seltz and H. Gruener examined the nasal mucosa.

TABLE 6 — *Analysis of two prescriptions bought from reputable pharmacist*

Normality of Nitrohydrochloric acid, dilute, bought on prescription	U.S.P.	1.9
For comparison		
Normality of Hydrochloric acid, dilute	U.S.P.	2.74
or 1.45 times that of the purchased sample of dilute nitrohydrochloric acid.		
1 cc. dilute nitrohydrochloric acid is equivalent to .16 grams or 2½ grains sodium bicarbonate.		

In the total metabolism of the human, minerals play an important part. Much is known about their specific rôles and the chemical processes involved. It would be too time consuming to give even a mere outline of this, but I would like to discuss a few phases of calcium metabolism.

Calcium occupies a peculiar position among the inorganic metabolites of the body. Calcium carbonate and phosphate form the rigid supporting structure of the osseous tissue, calcium salts are precipitated in certain pathologic conditions in other tissue and organs. Calcium has a peculiar influence upon the excitability of the motor system of animals, it is indispensable for the normal clotting of blood and it is distinguished from sodium

and potassium by the fact that the calcium ingested with food is excreted predominantly by the bowel, only a small part appearing in the urine. Ordinarily, in the adult, 65 per cent to 75 per cent if ingested calcium passes out in the feces.

Absorption of calcium is, in general, promoted by factors which tend to keep calcium in solution in the intestine. If a disproportionately large amount of phosphate is fed, the fecal calcium loss increases because more of the insoluble calcium phosphate is formed. On the other hand, anything which tends to promote an acidosis or to provide a more acid medium in the small intestine exerts a favorable influence on calcium absorption because it transforms the insoluble salts into soluble salts. Calcium absorption apparently takes place chiefly in the upper intestine where the reaction is relatively or actually acid.

It is not easy to raise the concentration of serum calcium far above normal by feeding calcium salts or high calcium containing foods. Intravenous or subcutaneous injection of calcium salts in amounts ordinarily prescribed is futile. If at all used, they should be given in very large doses — 5 grams four or five times daily — about four hours after meals or one and one-half hours before meals — this being necessary because the small intestine is least alkaline at this time.

The work of Sherman and others shows that a minimum intake of 0.45 gram of calcium fills the actual daily requirement for an adult. Many articles have been written treating of the deficiency of the American dietary, but a recent contribution by Sherman states: "When the results of 224 presumably typical American dietary studies were calculated to the usual basis of nutrition per man per day, the average protein content was found to be 106 grams or 140 per cent above the indicated maintenance requirement of 44 grams, while the average calcium content was 0.74 gram or 65 per cent above the acutal minimum of 0.45 gram." However, when there was a deficiency in the diet in the 224 cases the deficiency in calcium was six times more frequent than the deficiency in the protein. Therefore, when there is a deficiency diet it must be understood that the greatest deficiency occurs in calcium. To obviate this when minimum diets are given, milk should be the major portion of the diet.

Ullman feels that a high calcium intake is necessary and states that the relation of calcium to the other anions, such as sodium, potassium and magnesium, has been overlooked. He,

therefore, recommends a high calcium, low sodium chloride diet. He further contends that calcium absorption is inhibited in the presence of Na, Mg, and P, and that the anion sodium is responsible for the dehydration of the diseased tissue. Furthermore, he states: "Every acute congested condition, with which we always have to deal in acute as well as chronic sinus diseases, will react markedly if by restricting the intake of sodium chloride the dehydrating component of this measure is brought to the maximum. In other words, the stricter the salt restriction in acute infections and swellings, the better the result. For less acute and chronic cases the restriction of sodium chloride will not be as effective by its dehydrating power alone but will act through the relation of sodium to calcium."

The knowledge of the metabolism of magnesium and potassium is too meager and controversial to draw any conclusion about their rôles in disease. On the other hand, much is known about sodium chloride metabolism, and especially in relation to edema, but as yet no controlled evidence has been advanced to show that alterations of sodium chloride metabolism affect localized areas.

The reciprocal relation of sodium and calcium in the body is little understood. It would seem that before we can talk about this factor of the diet, less polemical information must be obtained about calcium inself. On the other hand, much is known about the fate of sodium chloride in the body and only quantitative metabolic studies will place this type of therapy on a rational basis.

In conclusion, one can see:

1. That there are fairly definite automatic regulatory chemical functions in the normal adult.

2. That it is unlikely that variations of diet affect the p_H of the blood and only slightly affect the CO_2 combining power.

3. That the diets as advocated should base their efficacy on some other action rather than alterations of acid base equilibrium.

4. That if one feels that certain nasal conditions are related to acid or alkaline tissue reactions, that large doses of NH_4Cl or $NaHCO_3$ should be used.

5. That the average mixed diet contains more than the minimum calcium requirement.

6. Finally, that possibly the diets as advocated will help fill the graveyard of the other deceased forms of treatment of obstinate nasal conditions.

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DISCUSSION

DR. FRANCIS B. BLACKMAR, Columbus, Ga.: I wish to thank the essayist, Dr. Striker, for bringing up this subject, in which many of us are interested. I feel that we get more good from those who do not agree with us than from those who do. If everybody agreed with us, we wouldn't get anywhere; our interest would die, and progress would be lost.

As Dr. Striker says, there are basal requirements for sodium and potassium, calcium and phosphorus, iron and sulphur. The dietician is concerned with the amount of these basal requirements. We as clinicians wish to know what are the recognizable effects of deficiencies and excesses in these factors in order that we can recognize them in our patients as parts of the diseases we already recognize. Having recognized them, we wish to correct them with foods and drugs.

NOTE—I am indebted to Dr. M. Herzberg, pathologist, for some of the laboratory work, and to Miss Peterson, dietician of the Jewish Hospital, for supervising the special diets.

I challenge each of you to ask those authorities in whom you have confidence to detail to you the clinical changes resulting in patients due to excesses and deficiencies in sodium, potassium, chlorides, hydroxides, carbonates, iron, sulphur, calcium and phosphorus, so that you can recognize these conditions or at least know when to suspect their presence. We rhinologists spend the majority of our time studying ambulatory patients. How do these factors show themselves in ambulatory patients? More especially, how do they affect the noses of these ambulatory patients? You may be told that excesses and deficiencies in these factors do not occur in ambulatory patients, but we feel that they do frequently occur. Dr. Shea called our attention to the fact that a sodium deficiency is a common cause of summer colds, following excessive perspiration. Dietary correction is too slow for them. They wish immediate relief. Their pale septa, nasal obstruction, sneezing, and watery discharge are often immediately relieved by a teaspoonful of salt. But sodium loss by the body is closely connected to suprarenal physiology. In fact, furnishing sufficient sodium has succeeded in maintaining life and health in dogs at the Mayo Clinic following complete removal of the suprarenals.

The many factors taking part in the several bodily equilibria can be grouped into nervous factors, chemical factors and glandular factors. All three groups interlock. Thus sympathetic stimulation causes suprarenal activity and, as stated above, suprarenal activity is closely related to sodium metabolism. Thus we group these factors on one side.

Dr. Striker objected to the idea that these factors produce their effects through shifts in the acid base equilibrium only, and we all agree with him heartily, for this is only one of many equilibria. Yet the acid base equilibrium is certainly one of the chemical equilibria and certainly acidity is related to sympathetic activity, suprarenal activity, and sodium metabolism. My article published in the "Laryngoscope" outlined those factors under the title of "The Autonomic Level." Since all these factors were mentioned, it was understood that all were included. This was objected to, and so I suggested a better name for the resultant of these nervous, chemical and glandular factors, namely, the N.C.G. level — N. for nervous, C. for chemical, and G. for glandular.

Objection has not been offered to the grouping of these various factors into two opposite groups. Objection is offered to the color of the nasal septum as an indicator of the N.C.G. level. Dr. Striker says it is of no value to him. I sympathize with him, as I can easily see how a rhinologist with constant inspection of the nose in the same light could more easily recognize color changes and be guided by them. For five years the Jarvis group have trained themselves to observe and interpret these color changes, and we find this information more and more helpful.

Years ago, an isolated doctor, who is with us here today, observed these same clinical pictures, with the red and pale septum, and used many of the same agents for shifting the color of the nasal septum. He published this twenty-five years ago. Then Dr. Stucky independently observed the same changes, and now fifty men for five years continue to confirm the observations of Dr. Kirkpatrick and Dr. Stucky, finding that it helps them in the treatment of their patients. It is not to be expected that we will

all find the same tools fitting our hands. Other indicators of the N.C.G. level are the basal metabolic rate, and, as Dr. Striker says, the urine acidity. None of these are free from error and they often contradict each other, but the position of the N.C.G. level is important. So let us each use the indicators we like best, but let us do our best on each patient to get some idea of the N.C.G. level and try to correct any imbalance, the existence of which we have reason to suspect.

DR. IRA FRANK, Chicago: I am sure that I speak in behalf of all those present when I acknowledge a debt of gratitude to Dr. Striker for his interesting and valuable observations. To many of you who, like myself, have been impressed with the value of diet therapy, Dr. Striker's conclusions must hold an especial interest.

The relation of diet to respiratory infections has been studied clinically for years. The general impression has been that a liberal diversified diet is of value in overcoming this type of infection. Such a diet is also of value in preventing infection because it is conducive to good health; and most bacteria cannot invade healthy tissue.

The diet that is most conducive to good health must usually contain liberal amounts of milk, fruits and vegetables. These are the foods that contain a preponderance of alkali elements; and there was a time, before we knew much about accessory foodstuffs, when the virtue of these foods was ascribed largely to their mineral content. That these foods are valuable health aids has never been seriously doubted. Investigators have merely wanted to know why they are beneficial.

Dr. Striker has been concerned chiefly with the physiological acidity or alkalinity of foods. This is very important; but it is, after all, only one property of foods. Our bodies can cope rather successfully for a long time with foods that are not physiologically neutral. Special mechanisms to which Dr. Striker has referred are present for this very purpose; but our bodies can not cope successfully with quantitative deficiencies. Deprive a man of the essential minerals or vitamins or an adequate amount of proteins and he will eventually became sick. Or overload him with nutrient material and force him to catabolize two or three times as much food as his tissues are able to handle easily, and he will become sick.

We must not forget that we all weighed about 7.5 pounds when we arrived and most of us now weigh more than 150 pounds. We have worked, planned, struggled to make a place for ourselves and families, maintained a definite body temperature under fluctuating climatic conditions, overcome the ravages of invading bacteria, sympathized, hated and loved. Everything that we now are came originally from our foods, and the energy necessary to do everything that we have done also came from these same foods. The ease with which we have come through life depends upon many things; but food plays an important rôle. We might have had rickets early in life, because of a lack of Ca, PO_4 and vitamin D in our diets. We may have had all of the children's diseases interspersed with a procession of respiratory infections, all due to a lowered resistance which, in many cases, is due to inadequate nutrition. We are none of us

overly intelligent; but we may have had abnormal difficulties acquiring knowledge and we may find it difficult even now to grasp things rapidly and with assurance; and much of this may be due to a lack of vitamin B in the diet of our mothers, as suggested by the well-known work of Dr. Siegfried Maurer of the University of Chicago, who, in a series of experiments with rats, showed that a lack of vitamin B in the diet of pregnant and nursing mothers affected the reasoning power and resulted in mental defects in their young.

Put in a maze, in the center of which was an appetizing piece of bacon, the animals whose mothers had been deprived of vitamin B took a much longer period to find their way to the tidbit than their fellows in the other cages whose mothers had been normally fed.

As children, we may have had many toothaches due to dental caries and the gingivæ may have been somewhat inflamed at all times; but this did not seem serious. Everyone else seemed to suffer in much the same way. As time went by, the teeth became loose, pockets formed, pus oozed out from around the teeth; and now we may be chewing on a full quota of porcelain teeth. Tooth decay and gingivitis are, to a large extent, symptoms of malnutrition. The color and quality of the mucous membranes of the gum tissue are indices of a vitamin C deficiency, barring mechanical factors. This leads me to wonder about the relation of diet, particularly of vitamin C, to the color and state of health of the mucous membranes of the throat and of the nasal septum, and I would like to ask the essayist to consider the following facts:

1. Isn't it generally conceded that the color of the mucous membrane of the nasal septum is an index to the state of health of the membrane and to the ease with which this membrane or others in the respiratory tract can be infected?

2. Isn't there a relation between the color of the membrane in a person who is not afflicted with a respiratory infection, and the blood pressure? I have in mind the facts that people who eat excessive amounts of protein are apt to have high blood pressure, whereas those who are vegetarian faddists are apt, in time, to have a low blood pressure. It seems to me that I see more people whose nasal mucosa is very pale because these are the people who suffer the most. A poorly ventilated tissue cannot be healthy and should be easily infected.

3. I should also like the doctor to consider what effect vitamin C, in adequate amounts, has upon the color or state of health of the mucous membrane of the nose and throat. By an adequate amount I mean about 250 mgs. of ascorbic acid per day, an amount to be found in a pint of orange or lemon juice, a pound of cantaloup, eleven ounces of raw cabbage, or a quart of tomato juice. I assume, of course, that the patients are also receiving other foods in adequate amounts. In short, what effect does an ample, diversified diet have upon the incidence and severity of respiratory infections? I ask the question advisedly because I am assured by many of my medical friends that people suffer less from respiratory infections when they are placed on diversified diets that contain an ample amount of foods rich in vitamins. People usually

suffer less from respiratory infections during the summer months. May this not be due to the abundance and cheapness of the high vitamin foods during this period? Is it not also true that people are less apt to be sick during the summer months, and may this not also be partially due to the more abundant consumption of vitamin rich foods during this period?

In closing, let me leave a thought with you that I have found to be most helpful. This is the era of intrinsic food analysis. We are apt to think of foods in terms of calories, proteins, carbohydrates, fats, minerals, alkalinity or acidity and vitamins. We must not forget that a well-prepared beefsteak is more than an assortment of nourishing chemicals, and that a glass of tomato juice is more than a few milligrams of vitamin C. Foods contain many substances whose presence is not even suspected and whose nature is not understood. But a potato continues to be a potato, and it has a certain dietary value because it is a potato and not simply because it yields an alkaline ash or because its skin contains vitamin B. We must endeavor, first of all, to determine the relation of foods, as foods, to general health and to specific disorders; and this can best be done by making observations on people. Members of the medical profession, regardless of specialty, are in an ideal position to make such observations. It is our privilege, in fact it is our duty, to make such observations and to promulgate the results and conclusions. The medical profession is dedicated to a life of service. Members of this profession are the chief counselors of the human race. People want to stay healthy; and good food is an aid to health. People don't know what to eat. Food faddists and charlatans sway the public with oratory. It is up to the medical profession to counteract oratory with facts and authority. It is our job, and I hope we shall all follow the good example set by our colleague, and by hundreds of other scientists, and study the relation of diet to health and disease.

Dr. Cecil Striker, closing: First, I wish to thank Dr. Blackmar and Dr. Frank for discussing my paper.

I want it understood that I am not a nose and throat man, so that I am not qualified to speak on the color of the nasal mucous membrane. I happen to be interested in diseases of metabolism, so it isn't that I have any difference of opinion with you; it is that some other nose and throat men do not have the same opinion you do. All I did was to control the dietary end, and the observations were made not by me, because I wouldn't be qualified, but by Dr. Gruener and Dr. Seltz. Incidentally, they were made on a code so that neither one of them knew what the other was doing, and I didn't know. The diets were all arranged so there couldn't be any possible subconscious cheating, as it were.

In regard to Dr. Frank's question on the state of health and the color of the mucous membrane, I can pass that by saying I am simply not qualified to answer that because I am not a nose and throat man.

In regard to the second question, that is, the effect of diet on blood pressure, I think that all of us are in accord with present attitude that we have toward diet in relation to blood pressure in nephritis. If one picks up an old textbook, he will find that protein is one of the most hazardous foods to take in. If any of you heard Dr. McLester's presidential address at the American Medical Association or read his article in the American Medical Association Journal, you found that protein is not very innocuous. It is a very necessary attribute. So the internists as a whole, I think, are not laying stress on the effect of high protein on blood pressure in nephritis. I think the greatest stress is laid on the caloric end now, rather than on the protein end.

In regard to the third question, the question of vitamin C, I don't know enough about that to answer the question. I will simply say I can't answer it. Thank you very much.

ANIMAL EXPERIMENTATION ON HEARING; ITS CLUES TO THE PREVENTION OF DEAFNESS*

M. H. Lurie, M.D.

BOSTON

During the past four years at the Harvard Medical School we have been studying hearing in animals (guinea pigs and cats), as a result of the discovery of the Wever-Bray phenomenon in 1930. Their phenomenon, as originally described, reported that when an electrode was placed on the auditory nerve of a cat and then connected with an amplifying apparatus of the radio type all sounds sent to the animal's ear through air were picked up in the auditory nerve as electrical currents. These currents, when amplified and sent through a loud speaker, gave back the original sounds and words sent in.

At Harvard, in the course of our experiments, we encountered a number of animals with definite pathological conditions.

1. Middle ear deafness.
2. Nerve deafness.
3. Deafness similar to otosclerosis.
4. Complete deafness of the inherited type.

All these animals have been studied with reference to the electrical response obtained from the cochlea and sections of the latter examined histologically. The correlation of these results points very definitely to the following facts:

1. Chronic progressive middle ear deafness can be due to neglect of the acute secretory or catarrhal ears of childhood.

2. Continuous exposure to constant noise of sufficient loudness will cause deafness of the so-called nerve type.

* From the Departments of Otology and Laryngology, Harvard Medical School, Boston, Mass. Research grant by Council of the American Academy of Ophthalmology and Otolaryngology, American Otological Society, and Josiah Macy, Jr., Foundation.

3. Toxic deafness caused by drugs or disease (such as typhoid, influenza, mumps) is probably due to a degeneration of the external hair cells of the organ of Corti.

In our experiments a group of animals, chiefly cats, gave electrical responses typical of those audiograms associated with middle ear deafness, i.e., loss of low tones with the high tones near normal. The animal with the slightest loss showed that adhesions in the region of the stapes may be present without any gross evidence of abnormality in the drum or middle ear. The animals with the greatest loss were those with the greatest histopathological changes of the middle ear. It is the study of this type of middle ear deafness found in animals which indicates the probable cause of chronic progressive middle ear deafness of the human adult. The least pathological change found was that of fibrous tissue filling the crux of the stapes and binding the stapes to the oval window, the stapes being able to move, as the joint was not involved. As a result the hearing of this animal was raised for all low tones up to about 2,000, higher tones coming through fairly normally. The rest of the middle ear structure, drum, malleus, incus, were normal. From this type of middle ear condition there was a progression to the middle ear being completely filled with granulations, adhesions and fibrous tissue and causing a marked deafness. The organ of Corti was normal in all of these animals.

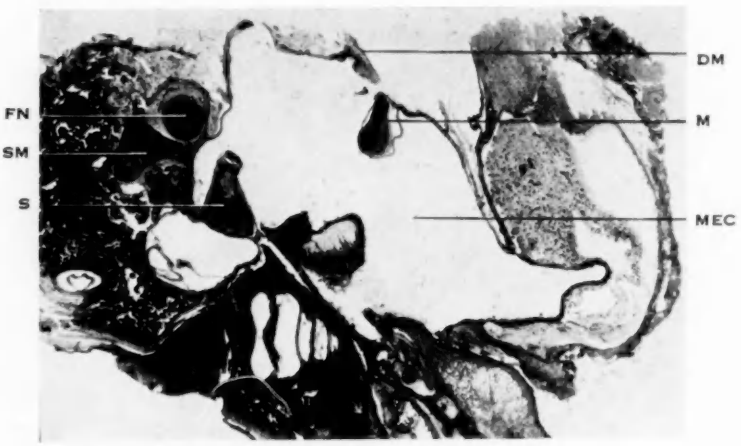

FIG. 1 — (Lurie) — Cat. Middle ear × 13. No evidence of active infection in middle ear cavity. Stapes filled with fibrous tissue.

FN = facial nerve
SM = stapedius muscle
S = stapes
DM = drum membrane
M = malleus
MEC = middle ear cavity

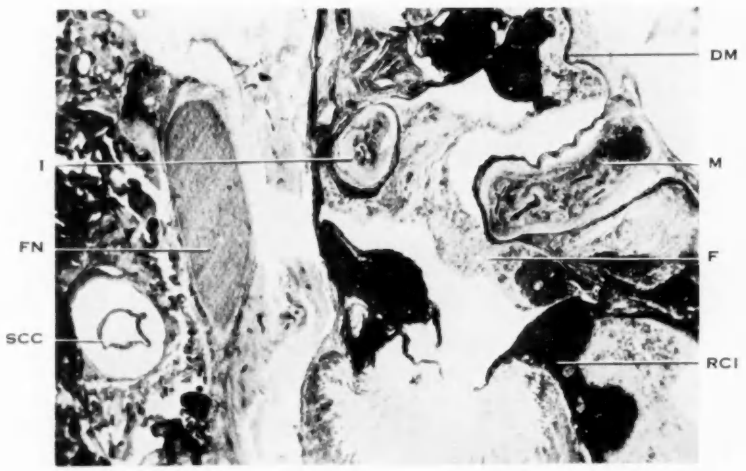

FIG. 2 — (Lurie) — Cat. Middle ear × 29. Epitympanic space. Round cell infiltration, and fibrous bands filling middle ear cavity.

I = incus
FN = facial nerve
SCC = semi-circular canal
DM = drum membrane
M = malleus
F = fibrosis
RCI = round cell infiltration

FIG. 3 — (Lurie) — Cat. Middle ear × 29. Tympanic cavity. Stapes filled with fibrous tissue. Tympanic cavity filled with round cell infiltration and fibrous tissue. Drum normal. No evidence of infection or discharge in external auditory canal.

S = stapes
V = vestibule
DM = drum membrane
RCI = round cell infiltration
M = malleus

Applying these facts to the human, it is very easy to reconstruct the pathological changes occurring in the middle ear which lead to chronic middle ear deafness. It is my belief that this condition can be the result of negligence or lack of appreciation of the importance of treating and handling properly the acute secretory and catarrhal ear of childhood. This is a condition to which little or no attention has been paid by the physician, patient or parent. The child recovers from the acute condition present, but the after effects may not be apparent until adult life. In all acute catarrhal or secretory ears there is a secretion of serum as a result of the low grade infection present. If the eustachian tube opens early, then the fluid is absorbed and there are practically no pathological changes following. When the tube remains closed for a long period, probably two weeks or more, there is absorption of part of the serum present, and an ingrowth of connective tissue into the remainder. The fluid will tend to stay in the region of the stapes, epitympanic space and the round window on account of capillary attraction. With the organization of this exudate, there are developed soft elastic adhesions which interfere very slightly, or not at all, with the child's hearing. However, as the child grows older, these adhesions tend to contract more and more, with the resulting increase of deafness, and a practically hopeless prognosis for the cure or arresting of the disease. It is these cases seen in practice, with drums slightly retracted, or thickened, eustachian tubes wide open and even with movement of malleus and drum under the Segalscope, that have as their underlying pathology adhesions of the stapes in the region of the oval window. The loss of high tones comes, in these cases, comparatively late and is due to a late degeneration of the organ of Corti.

That acute suppuration of the middle ear can cause this condition has been accepted by all. That a similar deafness can be caused by acute secretory or catarrhal ears, long after the acute condition has passed, has been recognized by but few. The repeated attacks of acute secretory ears in childhood should be regarded and treated as a serious condition not of the immediate present but because of the future damage that may occur.

From experimental animals in which deafness has been caused by exposure to loud tones, interesting results have been obtained. The work of Held, Yoshii, Wittmaack and others has been confirmed. There is definite degeneration of the organ of Corti. This degeneration is not localized to the region in

which theoretically the exposed tone is heard but is widespread especially toward the low tone range. We have obtained most marked degeneration of the organ of Corti in the middle of the basilar membrane. From this severe degeneration there is a gradation toward the apex and toward the round window. The degeneration in all our animals has been more toward the apex than toward the round window. The organ of Corti in these animals shows a very significant type of degeneration. The degeneration is in the external hair cells. In only one or two of the animals have we been able to obtain any evidence of internal hair changes and these only in a very localized area.

This type of lesion proves that the external hair cells are far more sensitive than the internal hair cells to sound and to degeneration. All animals so far examined with this type of degeneration, i.e., external hair cell loss, have had their thresholds raised 30 db. The threshold of sound for the external hair cells in some 30 db. lower than than for the internal hair cells, and this points to the probability that the internal hair cells have a different function than that of the external hair cells. From the evidence so far obtained, we believe that the internal hair cells have to do with the accurate discrimination of pitch and loud noises, while the external hair cells respond to tones and noises of very low intensity and are not concerned with the accurate analysis of the sounds so heard. The psychologists have shown from experiments on the human that with tones of minimum intensity there is poor discrimination of pitch, about one-third less accurate than with tones of increased intensity. This hypothesis of the dual function of the organ of Corti can explain why persons suffering from nerve deafness are deaf to spoken or whispered voice, but are still able to hear and analyze music so accurately.

It is also our belief that exposure to continuous loud noise which occurs in certain occupations, such as that of the aviator, boilermaker, riveter, etc., causes a degeneration of the external hair cells and does not involve the internal hair cells. If this is true, then the residual hearing of all deaf people can be explained by the functioning of the internal hair cells of the cochlea. We are now beginning a series of experiments to test this hypothesis.

Animals exposed to tones 2,500 dv. per second all showed definite degenerations as stated before. These animals were not only tested by the electrical method but also by the conditioned

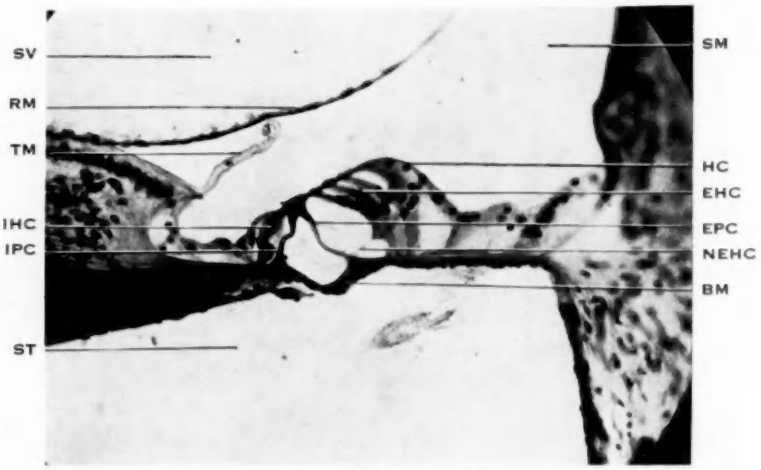

FIG. 4 — (Lurie) — Normal Organ of Corti. Guinea Pig × 300. Showing appearance of hair cells and their relationship.

SV = scala vestibuli
RM = Reissner's membrane
TM = tectorial membrane
IHC = internal hair cells
IPC = internal pillar cell
ST = scala tympani
SM = scala media
HC = Hensen's cells
EHC = external hair cells
EPC = external pillar cell
NEHC = nerve to external hair cells
BM = basilar membrane

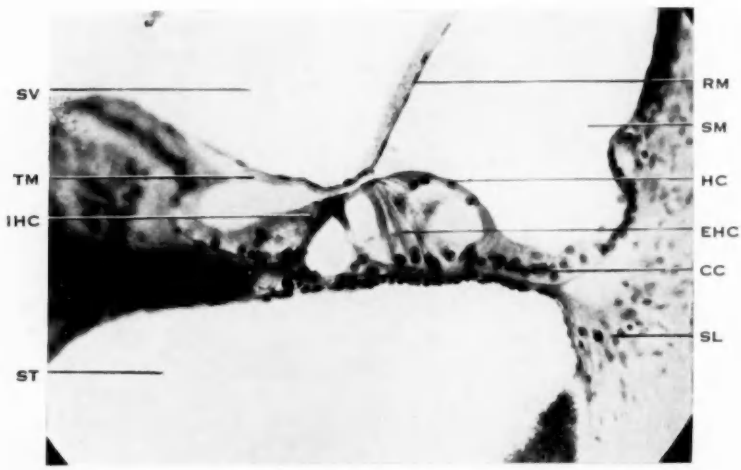

FIG. 5 — (Lurie) — Organ of Corti. Guinea Pig × 300. Degeneration of external hair cells due to exposure to 2,500 dv. for long period. Nucleus of internal hair cells is out of focus. External hair cells show complete degeneration.

SV = scala vestibuli
TM = tectorial membrane
IHC = internal hair cells
ST = scala tympani
RM = Reissner's membrane
SM = scala media
HC = Hensen's cells
EHC = external hair cells
CC = Claudius cells
SL = spiral ligament

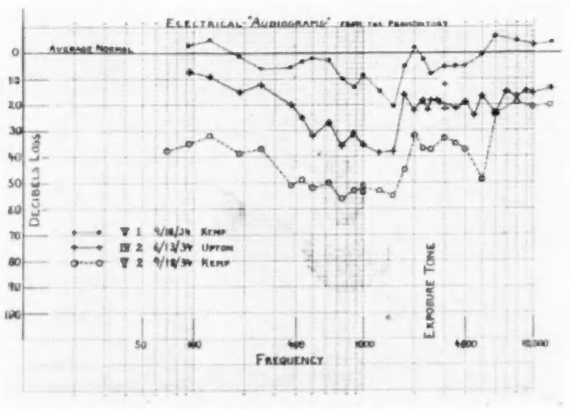

FIG. 6 — (Lurie) — Electrical Audiograms of Guinea Pig exposed to 2,500 dv. for various periods of time. Greatest loss of response below 2,000 dv. Also shows loss toward lower tones greater than toward higher tones.

reflex. The two methods agree very well and the lesions occurring in the cochlea agree with the localization of tones as has been reported (in May, 1935, at the American Otological Society meeting).

This was confirmed unwittingly by one of our co-workers who had exposed himself to the same tones to which the conditioned guinea pigs had been exposed. His audiogram taken before exposure was normal. Five months later he was tested again and the audiogram showed a loss of hearing similar to that of the guinea pigs exposed. A month later, after preventive measures had been taken, a third audiogram was found to be normal.

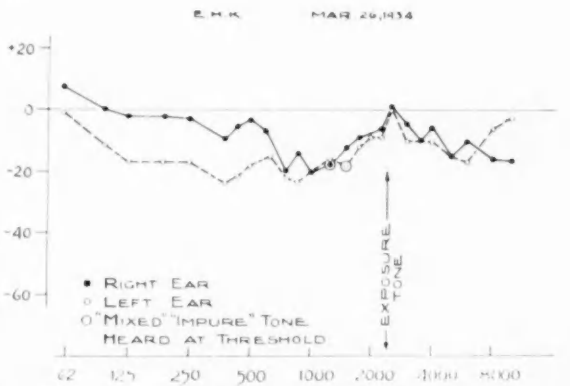

FIG. 7 — (Lurie) — Audiogram. Human ear exposed to 2,500 dv. per second shows similar loss as guinea pig exposed to same tone.

In one animal (cat), the electrical response showed a lesion similar to otosclerosis. All tones showed a loss for about 30-40 db. Examination of the ears showed new bone growth in the capsule of the cochlea, but the stapes was not involved. The lesion of the organ of Corti was the same as that of the animals deafened by stimulation. The external hair cells were degenerate and the internal hair cells showed practically no changes except near the round window where they also showed degeneration. On comparing the two organs of Corti from

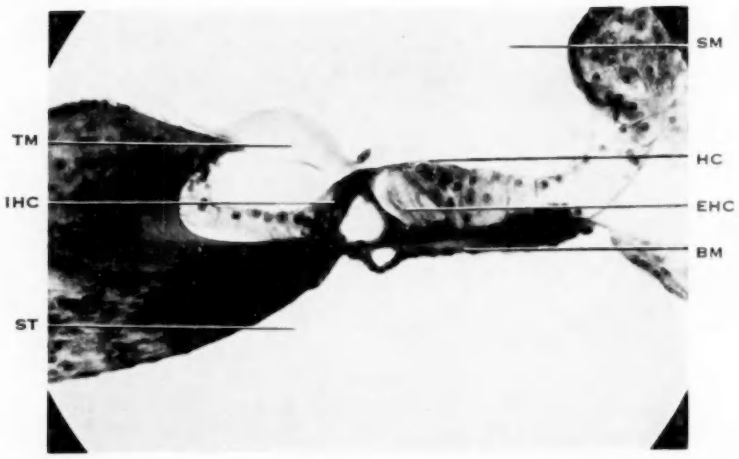

FIG. 8 — (Lurie) — Organ of Corti. Cat × 300. Degeneration of external hair cells. Internal hair cells appear normal.

TM = tectorial membrane
IHC = internal hair cells
ST = scala tympani
SM = scala media
HC = Hensen's cells
EHC = external hair cells
BM = basilar membrane

the stimulated deaf guinea pig and the otosclerotic cat, one could not tell them apart, except for the appearance of the spiral ganglia. The spiral ganglia of the cat showed degeneration of the cells, those of the guinea pig did not. (I do not doubt that if we waited long enough the ganglia cells of the guinea pig also would have degenerated). Toxic deafness whether due to drugs or disease in all likelihood should show similar lesions to those that have been described. Experiments to test this are now being carried out.

The similarity of the lesion and the audiogram thus described throws some light on the interpretation of hearing tests. Bone conduction loss of the so-called nerve deafness can be

explained as a degeneration of external hair cells in the organ of Corti and may be an indication of the amount of degeneration present in the organ of Corti. The increased bone conduction of middle ear deafness is the approach of bone conduction time toward normal air conduction time showing that a normal organ of Corti is functioning in the cochlea. Bone conduction, therefore, should be more accurately determined in all cases of deafness, for it may be a very definite clue as to the extent of the lesion present.

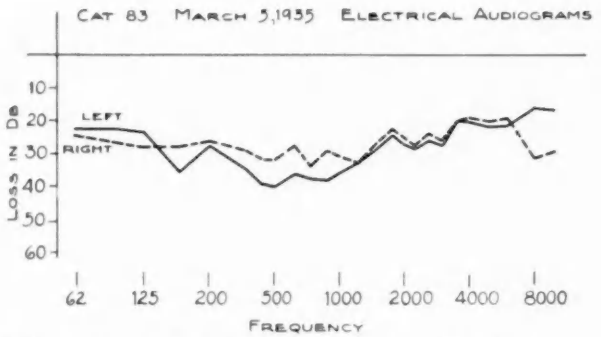

FIG. 9 — (Lurie) — Electrical Audiogram. Cat of Fig. 8. Average loss of 30 db. throughout the tone range tested.

Explosions or the so-called telephone shock can cause deafness, not only by disorganizing the organ of Corti, as S. Guild reported in 1919, but by actually tearing the organ of Corti from the basilar membrane. In experiments to prove that the basilar membrane vibrated, a guinea pig was exposed to an extremely loud tone of 400 cycles for about 5 minutes. After the exposure the animal's hearing dropped between 60-100 db. The cochlea when examined showed that in the upper turns the organ of Corti had been torn away from the basilar membrane and thrown into the scala media. The tectorial membrane and Reissner's membrane remained in their normal position. The basilar artery was not ruptured as there was no gross hemorrhage present in the cochlea. The prognosis, therefore, in all cases that have lost hearing due to exposure to loud and intense explosions should be very guarded as this type of lesion may be present.

Hereditary deafness, such as in the deaf mutes, is the result of lack of development of the organ of Corti and the cochlear nerve. In these cases the lesion present may be a complete

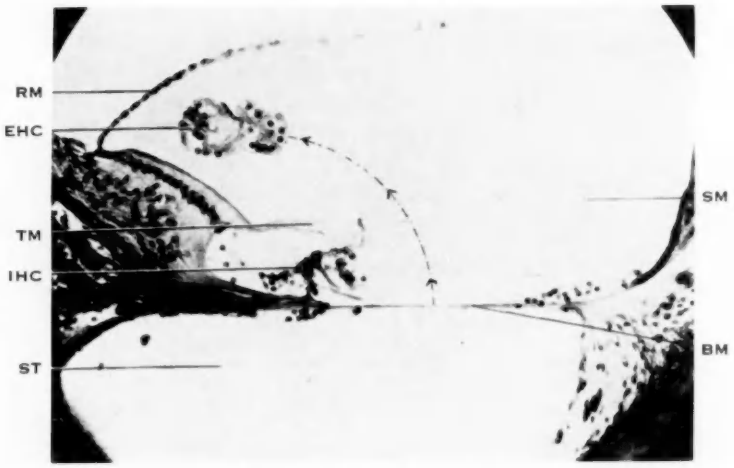

FIG. 10 — (Lurie) — Organ of Corti. Guinea Pig × 200. External hair cells and supporting cells, torn loose from basilar membrane, lying free in scala media. Note normal appearance and relationship of Reissner's membrane, tectorial membrane and basilar membrane.

TM = tectorial membrane
EHC = external hair cells and Hensen's cells
IHC = internal hair cells
ST = scala tympani
SM = scala media
BM = basilar membrane
RM = Reissner's membrane

absence of the organ of Corti with obliteration of the scala media as was found in one albino cat, or may show the changes that occur in the waltzing guinea pig. Here there has been a definite attempt by the animal to develop a normal organ of Corti usually in the last turn. The rest of the cochlea shows no organ of Corti that could be recognized as such.

Thus, it becomes the duty of the otolaryngologists not only to recognize the acute secretory ear of childhood, but to emphasize to the parents the importance of proper care and treatment to prevent the deafness that may become evident years after this neglected condition had been forgotten by the parents and the patient.

Patients should be warned that prolonged exposure to tones or noises of loud intensity is a very definite cause of deafness, and preventive measures should be used.

The dangers of toxic deafness, due to disease or drugs, should be recognized and prevented if possible. The excessive use of quinine, emetin, the salicylates should be guarded against and drugs giving the same therapeutic results without the toxic effect on the organ of hearing should be used as far as possible.

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DISCUSSION

DR. HARRIS P. MOSHER, Boston: This short paper is startling in the number of observations which it contains. Seldom has an ear paper contained so much that is new — and, I almost said, so much that is controversial. It takes two to make a fight, and I for one do not know enough about this subject to start one if I wished to. There is an air of positiveness about the writer's statements which is a bit disturbing. Naturally, I hope that they may be proved to be true, because Dr. Lurie is one of our infirmary group and we are shining by reflected light.

The beginning and the end of the paper are mild enough. But the main body of it is full of explosive deductions. Taking the paper as a whole it is a most important contribution.

First, as to his conclusions: He advises the early recognition and treatment of acute secretory ears, stating that both parents and otologists do not realize the progressive nature of the adhesions which follow in these cases. I, for one, would like to go on record as having recognized it. In fact, I think that most of us have, and that Dr. Lurie overstates the lack of recognition.

The second conclusion is that patients should be warned of prolonged exposure to noise. He has a pretty experiment to illustrate this point. He does not go quite so far as to advise that no one should live in cities.

His third conclusion is that drugs which cause toxic deafness should be avoided. This, of course, has been known for a long time.

These conclusions are not startling. In fact, they are rather old. What Dr. Lurie, however, has done is by animal experimentation and animal findings to give proof of these conclusions. Most of us have believed them for years, but have not had the animal findings to back them up. This accomplishment of Dr. Lurie's is welcome and important.

In speaking of secretory ears, he states that closure of the eustachian tube for two weeks results in adhesions. This seems to me rather too short a time.

Dr. Lurie makes the rather self-evident statement that the more pathology, the greater the loss of hearing. He states also that the height of the lesion is usually in the middle of the basilar membrane and that there is a spread of the lesion upward toward the low tones, and very little spread downward toward the round window. I was interested in Dr. Lurie's use of the word "spread." It came out in the papers read at Toronto this year, that most of the essayists believed that the hearing

for any given tone did not have a precise and definite localization, but spread over a considerable area. Dr. Lurie is making his pathology spread in the same fashion.

Before taking up the other theories laid down in the paper I should like to say a word about the testing of hearing in animals. I have had numerous conversations with Dr. Lurie on this subject. He feels that the conditioning of an animal to certain sounds is scientifically accurate. For your information and for mine, I should like to have him, in closing the discussion, give the method by which conditioning of the animals to certain sounds is accomplished. If he were using dogs, and especially my dog, I should think this might be accomplished easily, but I am a doubter when it comes to my cat.

I now come to the most important statement in the paper, from the standpoint of physiology, namely, that the degeneration found in the organ of Corti was in most cases in the external hair cells. These, the writer states, are more sensitive and their threshold of stimulation is 30 db. lower than the internal hair cells. The internal hair cells, he states further, determine the pitch and loudness of noise, whereas the external hair cells are more sensitive and do not give as accurate an analysis of sound. In other words, the organ of Corti has a dual function. To postulate a dual function to any organ is dangerous. Further, Dr. Lurie states he believes that as the external hair cells are the ones most often involved, the fact that a patient has residual hearing is due to the preservation and functioning of the internal hair cells. Boilermakers' deafness is stated to be due to pathological changes in the external hair cells. All the foregoing is important; in fact, very important if true.

I was interested in the happening to one of his co-workers who became deaf on account of exposure to high sounds. What interested me was the fact that his loss of hearing was not permanent because in five months his hearing was back to normal.

The writer of the paper holds that bone conduction loss in otosclerosis is due to impairment of the external hair cells. In other words, the loss of bone conduction is a measure of the amount of degeneration of the organ of Corti. He makes a plea for a more careful determination of bone conduction, using this as a measure of the amount of degeneration. This is an old idea put in a slightly new way. All of us have felt for years that an excessive loss of bone conduction meant nerve involvement.

What the writer says of telephone shock is of practical interest. He found that an animal exposed to an explosive sound suffered a disorganization of the organ of Corti and tearing of the basilar membrane. He stated that this experiment was to prove whether or not the basilar membrane vibrated. The experiment seems to me too violent to draw such conclusions. If a man is blown to pieces by dynamite, naturally his whole body vibrates.

It is interesting in this experiment that the tectorial membrane and Reissner's membrane remained intact. Shambaugh seems to be having a hard time with his tectorial membrane, unless he gets some satisfaction out of the fact that it cannot be blown up.

Coming back to the cat that had otosclerosis and a loss of external hair cells, I am wondering if Dr. Lurie has looked up the question whether or not there was any hereditary tendency in this cat. His final observation, namely, on the albino cat, that there is an obliteration of the scala media and that in the waltzing guinea pig the cochlea is only partly developed, are striking.

Dr. Louis K. Guggenheim, St. Louis: I spent a large part of last night with Dr. Lurie looking at photomicrographs and listening to him tell of his findings, and I want to express my intense appreciation of the interesting results of his research and of the excellence of the photomicrographs. I think it is a very outstanding result.

Nothing has been said concerning the underlying cause of hearing impairment from serous exudate into the tympanum. The tympanic cavity in its development is, at first, chiefly a potential cavity, consisting in the early embryo of an extension of the first pharyngeal pouch. Not until the fourth or fifth month does the mesenchymal tissue resorb sufficiently for us to recognize a tympanic cavity. Now the resorption of mesenchymal tissue is not always complete. Dr. Lurie has told you that serous exudate results in the formation of elastic fibers, then connective tissue about the stapes, which contracts and brings about definite and even progressive impairment of hearing.

This may occur; but more frequently, I believe, the mesenchymal tissue in the tympanic cavity is the underlying cause of the impairment of hearing. This tissue is quite soft and elastic, but when infection occurs after birth it may be converted into adult connective tissue.

Serous exudative conditions should be treated, of course, as best we know how. But regardless of treatment, impairment of hearing may occur, I believe, from the conversion of mesenchymal bands about the stapes into connective tissue.

In regard to the animal with otosclerosis-like changes, Dr. Lurie gave you the impression that otosclerosis is a disease of hearing. It is not a disease of hearing, but a condition in which the capsule of the ear shows an osseous dystrophy with sites of predilection. Hearing impairment is a complication but may be entirely absent. The osseous dystrophy in the animal presented does not seem to be otosclerotic.

However, the specimens are very remarkable, and I certainly have enjoyed the dissertation very, very much.

Dr. M. H. Lurie, closing: I wish to thank Dr. Mosher for putting some joy into the discussion, because I know exactly how you feel listening to me—as I feel when I listen to the psychologist or the physiologist when they are technical.

On the question in regard to acute secretory ears, I agree with Dr. Mosher. A number of us do know it, but we pay no attention to it. That is what I think is the nub of the matter. We make the diagnosis; we see

the patient; but seldom do we ask that patient to come back three or four times, and test the ears to make sure they are normal. I know I have fallen into that habit myself. I think most of us do.

In regard to the question of what happens pathologically, I simply pointed out the probable changes in the human and what may happen in the middle ear. I have tried to show that in animals we find similar conditions. Whether we can say they are absolutely the same in the human being, I do not know. I hope someone will find human specimens that will confirm this.

In regard to otosclerosis, I think the men studying this condition from a pathological viewpoint have made much of the bone pathology. They should also study the organ of Corti in otosclerosis. I know when I try to make a diagnosis of otosclerosis it is on the living, I do not care to make it after the patient is dead. It doesn't interest me any more. I want to know what is happening and what I can do for him when he is alive.

OBSERVATIONS ON CHRONIC INFLAMMATORY LESIONS OF THE TRUE VOCAL CORDS

GABRIEL TUCKER, M.D.

PHILADELPHIA

THE TRUE vocal cords play a most important part in the phonatory, expectorative, respiratory and protective functions of the larynx. During these activities the trauma incident to almost constant use of the cords is probably a factor in the development of pathologic processes in these structures of the larynx. Chronic nonspecific inflammatory lesions of the larynx, as well as neoplasms, benign and malignant, occur most frequently on the true cords.

Where a localized lesion exists on the vocal cords additional muscular effort is required in attempts at phonation. The lesion itself becomes the site of a local irritation and has a tendency in most cases to increase in size. Where contact of the lesion with the other cord is made we see evidence of localized irritation on the opposite cord and frequently the development of a lesion. Vocal nodules originate usually as a lesion on one cord. Later, at the point of contact on the opposite cord, a nodule will develop. Contact ulcer occurs as a bilateral lesion, possibly in the same manner. As the lesion increases in size, greater difficulty is experienced in phonation and increased muscular effort is demanded by attempts at clear phonation. This results in a vicious cycle, the overacting musculature producing greater irritation and progressive increase in size of the lesion, and the larger mass demands more muscular action and produces increased irritation to the point where aphonia and dyspnea may develop.

In the treatment of localized chronic inflammatory lesions of the true vocal cords it is necessary to break this vicious cycle. *The first* requirement is to restore the normal contour of the cord by removal of the tumor formation without injury to the cord itself. *The second* requirement is vocal rest. The overacting musculature still remains after removal of the tumor

and a period of vocal rest should be enforced until the surface of the cord is healed. When the cord is healed, reeducation in the proper method of voice production is necessary.

Where the lesion is early, absolute vocal rest without surgical removal of the lesion may effect a cure; but where a definite tumor formation has occurred this is usually not sufficient. Much time will be saved by scalping off the excess tissue and restoring the normal contour of the cord before the rest period is carried out. In cases where a diagnosis is in question, the removal of tissue affords the opportunity for correct diagnosis by histologic examination of the tissue.

Chronic irritation produced by the localized tumor mass on the cord may possibly be one of the factors also in the production of the change from a benign to a malignant tumor.

Occurrence of Benign Inflammatory Lesions

In the five year period from July 1930 to July 1935 there have come under my observation at the Bronchoscopic Clinics of the University of Pennsylvania at the University Hospital and the Graduate Hospital, 145 cases of chronic inflammatory lesions of the vocal cords classified as follows:

Vocal nodules	53 cases
Contact ulcer	9 cases
Inflammatory tumor	37 cases
Polypoid and redundant masses, involving the cords and ventricle (prolapse)	23 cases
Fibroangioma	14 cases
Leukoplakia	9 cases
Total Chronic Inflammatory Lesions of Larynx	145 cases

Etiology — The greatest factor in the development of chronic inflammatory lesions of the vocal cord is probably overuse and improper use of the voice. Excessive smoking, alcoholism, irritating vapors, constant inhalation of dust or irritating vapors, frequently talking against noise in a dusty place, all contribute to the development of these lesions. Probably the best example of the result of overuse of the voice is seen in the huckster. Intense voice strain from cheering at a ball game frequently produces a slight submucous hemorrhage on the cord which finally becomes a localized tumor mass as the blood clot organizes and healing takes place.

Location of Lesion — Chronic inflammatory lesions may occur at any location on the true cords. Vocal nodules occur at the nodal point at the junction of the anterior and middle third of the cord. Inflammatory thickenings occur most frequently on the upper surface and middle portion of the membranous cord. Where redundancy occurs it is usually seen as a flapping mass over the top surface of the cord projecting toward the median line. Keratoses may appear on any portion of the cord. Pachydermia laryngis occurs in the interior of the larynx, the greatest thickening being in the posterior portion of the larynx involving the posterior ends of the cords. The older descriptions of pachydermia include contact ulcer. Contact ulcer has been so clearly differentiated as a separate clinical entity by Dr. Chevalier Jackson that confusion no longer exists.

The term contact ulcer correctly emphasizes the chief etiologic factor, contact trauma. I have noted in one case the development of contact ulcer. A pedunculated mass had been removed from the anterior portion of the cord. This mass had been propping the cords apart and from efforts at voice production the patient had developed an overacting musculature in the larynx. When the mass was removed the vocal processes were brought together with such force that ulceration occurred on both sides where the vocal processes came in contact. Absolute vocal rest and reeducation in the correct method of voice production produced complete healing of the ulceration in this case.

Symptoms — The symptoms of chronic inflammatory lesions of the vocal cords do not differ from the symptoms of lesions of neoplastic origin. The first symptom is impairment of voice with vocal fatigue. Hoarseness may at first be intermittent, later becomes constant and, finally, with the advancement of the lesion, the patient may become completely aphonic. Local discomfort in the larynx may be felt. This is due probably to the increased muscular effort necessary for voice production rather than to the presence of the lesion itself.

Diagnosis — In any form of laryngeal disease a routine diagnostic procedure including careful history, general examination of the patient, x-ray examination of the neck, chest and swallowing function, and serologic examination should be followed. The object of these routine studies is to exclude or demonstrate any general or local extra-laryngeal disease that might have a definite relationship to the local lesion in the larynx.

Careful mirror examination and study will aid in determining the character and location of the lesion. This may be supplemented by direct examination if this is indicated. The final differential diagnosis is made by the histologic examination of the tissue. This can readily be carried out in many cases because it is necessary to restore the normal contour of the cord as the first step in treatment and the examination of the removed tissue will serve to establish a definite diagnosis.

Treatment — Absolute vocal rest, using a pencil and paper for all communication may be tried in every case. It is of special benefit where there is a generalized inflammatory reaction in the larynx. The use of alcohol and tobacco should be stopped. Local application should be by inhalation and the dropping of bland medication into the larynx. Caustic applications, especially nitrate of silver, should not be used.

Surgical Treatment — Where the lesion is localized and abnormal masses of tissue are present on the true cords scalping off these abnormal masses of tissue by means of direct laryngoscopy in order to restore the normal contour of the cord should be considered. A word of caution is necessary in giving this advice. Great care must be taken in removal of tissue that the true cord is not injured. A very careful technic is required. If the deeper structures of the cord are injured, scar formation will result and permanent impairment of the function of the cord will result.

Methods of Removal — My own preference is for the use of the anterior commissure laryngoscope and cupped forceps of Jackson. The individual operator, of course, should use the method by which he will obtain the greatest method of operative skill. A very gentle touch is required. The rule should be not to remove too much tissue. At another sitting further tissue can be removed if need be, but if too much is removed it cannot be replaced. A roughened elevated surface remaining after tissue removal will flatten out as the denuded cord heals and the normal contour of the cord will be restored in most cases. If small tags of tissue remain subsequent to removal of tissue after healing has occurred, they may be scalped off in the same manner as the first removal. Absolute vocal rest should be maintained until the cord is completely healed. Reeducation of the laryngeal musculature for proper tone production should then begin. The following cases with illustration are submitted.

CASE 1. *Vocal Nodules* — Patient, male, 56 years of age, engineer, referred by Dr. Karl M. Houser. He complained of early vocal fatigue and hoarseness in the afternoon. Hoarseness became constant as the condition progressed. Discomfort in the region of the larynx was noted after prolonged use of the voice. Routine examinations were negative. Mirror examination revealed well developed nodules with local inflammatory reaction on both cords. (Fig. 1A).

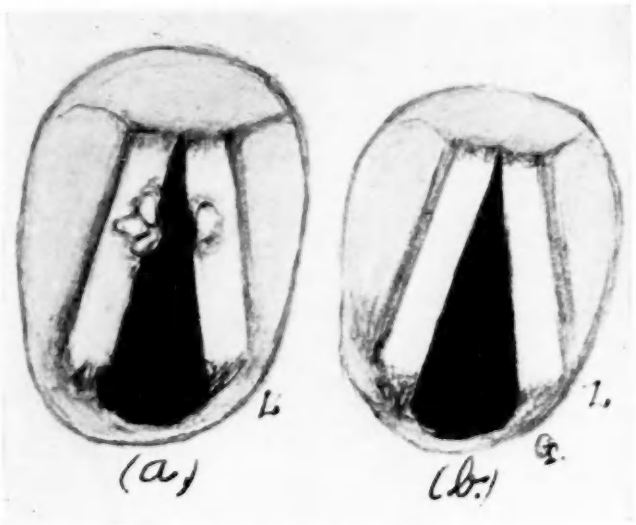


FIG. 1 — (Tucker) — Case 1.

A — Mirror appearance of the larynx showing bilateral vocal nodules with marked inflammatory reaction surrounding the nodular masses.

B — The same case one year after direct laryngoscopic removal. No recurrence, voice normal. Patient was allowed to use the voice six weeks after the direct laryngoscopic removal of the nodules and reeducation of the vocal mechanism.

Treatment — The tumor masses were scalped off under direct laryngoscopy. When Dr. E. A. Case examined the tissue he reported, "Tissue shows marked thickening of the squamous epithelium with edema, hemorrhage and cellular infiltration of the deeper tissue. No evidence of malignancy." Absolute vocal rest was enforced until the cords were completely healed. The patient was then instructed in the proper method of tone production. He cooperated well and at the end of a year examination showed the larynx to be normal (Fig. 1B), the patient having a clear voice.

Case 2. *Chronic Inflammatory Tumor of the Cord* — Patient, male, 42 years of age, archeologist, referred by Dr. William Wood, Philadelphia. He complained of hoarseness of over a period of two months duration, which followed a severe cold. He was a heavy smoker and a fairly moderate consumer of alcohol. Vocal rest had been attempted for several months prior to consultation. Routine examinations were negative. Mirror examination revealed generalized inflammatory reaction in the mucous membrane of the larynx. On the anterior end of the left cord, projecting into the glottic lumen, was an irregular

A B

FIG. 2 — (Tucker) — Case 2.

A — Mirror appearance of chronic inflammatory tumor involving the left cord.

B — Small granulation mass reformed after first direct laryngoscopic removal. The cord cleared after subsequent removals and at the present time is normal in appearance. Voice is normal.

tumor mass. On attempted phonation the redundant tissue lay on top of both cords, acting as a damper (Fig. 2A). The appearance was strongly suggestive of neoplasm, although no definite infiltration of the cords could be seen. It was decided to scalp off the redundant tissue by direct laryngoscopy. Removed tissue was reported by Dr. Case, "The tumor consists of loose edematous tissue covered with squamous epithelium. It is not malignant." Absolute silence was enforced, smoking was gradually stopped, alcoholics were discontinued. A small mass of granulation tissue

formed on the cord at the site at which the tumor was removed. The cord was not infiltrated (Fig. 2B). The tissue was again removed and reported as inflammatory. The patient then returned to his home in England where he was kept under observation by Mr. Lionel Colledge. Reeducation in the proper method of voice production was given. He returned at the end of a year and examination with a mirror showed the previously affected cord to be perfectly smooth with no scar formation. It was slightly redder, however, than the uninvolved cord. His voice was good. At this time Dr. William Wood found evidence of tonsillar infection and removed the tonsils. After the tonsillectomy the excessive redness in the cord disappeared. The patient returned to England where he is being kept under observation by Mr. Lionel Colledge.

Comment — The direct laryngoscopic removal of abnormal tissue from the vocal cord in this patient established a diagnosis of benign lesion and at the same time restored the normal contour of the cord. Whether or not further lesion will develop it is impossible to predict. It is not possible to do more than to restore a larynx to normal. The cooperation of the patient will have a great deal to do with the final result. Proper follow-up observation, however, will be continued.

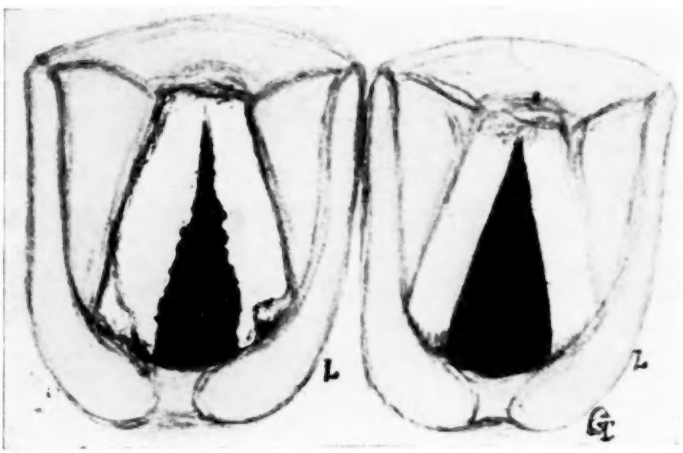
Case 3. *Redundant Inflammatory Tissue (Beginning Eversion of the Ventricle?)* — The patient, a male, 44 years of age, a private secretary, was referred by Dr. George B. Wood. On admission he complained of loss of voice, pain and soreness in the region of the larynx, strangling non-productive cough occurring at night when the patient felt that he was threatened with asphyxiation. The first symptom noted by the patient was hoarseness, which began with and continued after an attack of acute laryngitis two years previously. Mirror examination at the time of onset of his condition was reported to have shown edema of both vocal cords. Hoarseness from this time on had been persistent. The patient had developed frequent colds and on these occasions had become completely aphonic. Routine studies were negative. Mirror examination on admission showed thickening and irregularity of the left cord and a mass projecting apparently from the left ventricle. The redundancy was so great that the projecting mass came between the margins of the cord on attempted phonation, producing complete loss of voice.

Treatment — The redundant mass was scalped from the left side of the larynx. Care was taken not to injure the deeper structures of the cord. The right cord was not treated, although there was some tissue redundancy of the mucous membrane on its upper surface. The patient was placed on absolute silence and kept under observation. The denuded area of the cord healed satisfactorily, but there remained, however, slight irregularity of the anterior end of the left cord where removal of the redundant tissues had not been attempted. Subsequent removals were made at bi-weekly intervals. The tissue submitted for examination was reported by Dr. Case as, "Loose edematous fibrous growth covered with mucous membrane. It is benign." The right cord remained slightly thickened. The patient was allowed to use his voice and was so much improved that it was thought advisable to postpone any operative removal of tissue from the right cord for at least six months. At the termination of this period he had developed an excellent voice, cough was entirely relieved and the redundancy of the right cord had receded greatly, although there had been no operative work on this cord. The patient is still under observation by Dr. George B. Wood, with whom he was seen in consultation.

Comment — Direct laryngoscopic scalping off of the redundant tissue in this larynx relieved the patient's symptoms, although only the left side was operated upon. The right side improved because the irritation of the redundant mass of tissue from the left side had been removed. It is probable that further tissue from the right side will require removal.

Case 4. *Contact Ulcer* — The patient, a man 48 years of age, automobile accessory salesman referred by Dr. John R. Mench. On admission the patient complained of hoarseness of four months' duration. There was a feeling of a lump in his throat and a slight pain on swallowing. The hoarseness and pain had become so severe that the patient was forced to discontinue his work. He was a heavy smoker and a moderate user of alcohol. Routine studies were negative. Mirror examination showed the typical appearance as described by Dr. Chevalier Jackson as contact ulcer. The thickened tissue over the vocal processes of both arytenoids was scalped off by direct laryngoscopy. Histologic examination by Dr. Case, "Squamous mucous membrane, greatly thickened, submucous tissues are the

seat of chronic inflammation. No evidence of malignancy." The patient was placed on absolute vocal rest until the denuded areas had healed satisfactorily. After vocal training, he developed proper control of his larynx and was allowed to go back to moderate use of his voice. His larynx returned to normal. At the present time, two years after treatment, his voice is normal and he has no pain or vocal fatigue.



A B

FIG. 3 — (Tucker) — Case 5.

A — Mirror appearance showing thickened epithelium forming crusts entirely surrounding the glottis and fixing the vocal cords to such an extent that the patient was aphonic. Tissue removed reported as leukoplakia.

B — After repeated scalping off of the abnormal tissue patient's voice returned after two months' treatment and mirror examination of the larynx shows the cords to be practically clear except for a small area on the left cord. The voice is clear and there is certainly no evidence that this lesion is precancerous after three years' observation.

CASE 5. *Keratoses, Leukoplakia Laryngis* — The patient, male, 55 years of age, a laborer, was referred by Dr. Thomas Furlong, Bryn Mawr, Pa. On admission the patient stated that he had been hoarse for one year, following a cold. At the time of the examination he was completely aphonic. He was an occasional light smoker, used no alcohol. Routine examinations were negative. Mirror examination showed both cords to be entirely covered with a thick whitish tissue formation which extended subglottic anteriorly and posteriorly beneath the vocal cords and the vocal processes of the arytenoids (Fig. 3A). The motility of the cords was so greatly impaired that their margins would not approximate. The vibration of the cords was prevented by the thick crust-like formation over the entire upper

and inner surface of the cords. The neck showed no adenopathy. The provisional diagnosis of leukoplakia was made. The lesion was so extensive that if it had been considered pre-cancerous the only logical treatment would have been laryngectomy.

Treatment — Direct laryngoscopy was done and the thickened tissue was removed as a crust formation, leaving a raw bleeding surface, at three sittings. The local application of tincture of benzoin was made over the denuded areas, after each removal. During the past three years that the patient has been under observation there have been slight recurrences and slight removals of small areas of exudate with but very little crusting. At the present time his larynx shows a small whitish area, thickened, elevated tissue on the internal surfaces and anterior end of the left cord (Fig. 3B). The patient has a good voice. All removed tissue had been examined very carefully by Dr. Case and reported, "Marked thickening of the epithelium covering the cord. There is no evidence of cancer. Diagnosis: Leukoplakia of a marked degree."

Comment — In this case the removal of a thick crust-like formation of exudate and thickened epithelium restored the contour of the cords allowing them to approximate and after the raw surfaces had healed the patient developed a good voice. There is very little evidence of leukoplakia at the present time and certainly no evidence that the condition is undergoing malignant change after three years' observation.

Conclusions

1. Tumor masses due to chronic inflammatory disease on the true vocal cords that do not disappear promptly under absolute vocal rest should be removed surgically by scalping off the irregularities in order to restore the normal contour of the cord.

2. Superficial scalping off of tissue masses will leave a smooth surface which will be covered by normal mucous membrane when the lesion heals. Restoration of the normal contour of the cord will stop the vicious cycle produced by the presence and progressive enlargement of the tumor mass on the cord. Proper voice training will restore the voice and larynx to normal in many cases.

3. Attempted removal by cauterization, either thermal or chemical, of a tumor mass formation on the true vocal cord is

likely to produce sloughing with scar formation and permanent impairment of cord function.

4. Direct laryngoscopic removal with cupped forceps of the abnormal tissue is an accurate and safe method, in trained hands, of surgical removal of abnormal tissue masses on the true cords. The histologic examination of the removed tissue provides a definite basis for correct diagnosis of the character of the lesion.

DISCUSSION

Dr. Ellen J. Patterson, Pittsburgh: Dr. Tucker has covered the subject so thoroughly in his presentation that there is little to add by way of discussion, except to emphasize his remarks upon one or two points.

I would take exception to his remarks about the protective function of true vocal cords, as it seems to me the ventricular bands play a more important part in this function.

Almost every patient with disease of the larynx, whether due to inflammation or growth of the vocal cords, dates his trouble from a cold which never cleared up, as hoarseness is the most constant symptom.

It is important to examine thoroughly the nasal accessory sinuses, teeth and tonsils, because infection plays an important part in the etiology of many chronic inflammatory lesions of the vocal cords. A general physical examination is also important as we sometimes encounter chronic inflammatory lesions of the cords as local manifestations of a general condition.

In considering the etiology of chronic inflammatory lesions of the cords, it is of interest to note the findings of Negus (Negus, V. E.: *Mechanism of Larynx*, The C. V. Mosby Co., St. Louis, 1929, p. 539) in connection with his studies of the mechanism of phonation. He observed that:

"The cords move to the greatest extent at a point midway between the tip of the vocal process and the anterior extremity, while the arytenoid cartilages are motionless.

"With reference to the observation that the midway point of the membranous vocal cord is the point of maximum movement, there is an interesting corollary in the production of singer's nodules. These formations commence as submucous hemorrhages on the vocal cord of singers or speakers, usually those of the female sex.

"The nodes always occur at this exact spot, which is the point of maximum velocity. The force of impact of one cord against the other is, therefore, greatest here and it is easy to see that in powerful phonation a small submucous hemorrhage can be produced.

"Once a small area is raised above the surface it will receive additional injury at each vibration of the cords because of their impact with maximum force at this point; a vicious circle is thus produced, often with a large node as the result."

Those of you who saw the film on the larynx by Dr. Joel Pressman can appreciate Dr. Negus's findings, and the force with which the cords approximate in phonation.

In disease of the larynx, as in any other branch of medicine, though I think especially in diseases of the larynx, diagnosis is the question of paramount importance. Early diagnosis is important in any patient exhibiting hoarseness which does not clear up promptly or in a patient with recurring attacks of hoarseness.

Chevalier Jackson has taught us for years that anything which interferes with approximation, vibration or tension of the vocal cords will cause hoarseness. Also that every case of chronic hoarseness is a potential case of malignant disease and a diagnosis of chronic laryngitis should never be made until every other cause of hoarseness has been ruled out by elimination.

Indirect examination of the larynx for diagnosis of lesions of the vocal cords should be followed by direct laryngoscopy in all patients where it is difficult to obtain a satisfactory exposure of the glottis.

The larynx of any patient, even a new-born babe, can be examined by direct laryngoscopy. By direct laryngoscopy all parts of the larynx can be inspected — the anterior commissure, cords, ventricles, post-commissure, and even the subglottic area.

However, operative procedures upon the cords by direct laryngoscopy must be performed with extreme precision, otherwise the larynx may be irreparably damaged. In the removal of a specimen for histological study to confirm diagnosis in these cases, the tissue must be removed from the exact area involved; otherwise the pathologist may be confused in his diagnosis between benign and malignant tissue.

In the removal of chronic inflammatory lesions of the true cords by direct laryngoscopy, as in cases reported by Dr. Tucker, extreme caution must be taken to leave the cords intact, as any injury to them will damage their function permanently. Following removal of these lesions, the patients should be kept under observation for at least a year.

I think Dr. Tucker is to be congratulated on his presentation.

Dr. M. F. Arbuckle, St. Louis: Dr. Tucker's paper is very important because of the rather frequent occurrence of lesions such as he has described and the necessity for differentiating between these and malignant disease of the larynx. This often requires microscopic study.

In addition to the deleterious effects of alcohol, tobacco, dust, chemicals, and improper use of the voice, it must be remembered that in the presence of chronic suppurative disease of the upper or lower respiratory tract, the larynx is constantly exposed to infectious material. Furthermore, certain types of kidney disease and heart disease occasionally are accompanied by changes in the larynx. Of course, syphilis and tuberculosis must be kept in mind; and, at times, most careful study is required in order to make a decision regarding the presence or absence of one or both of these. Hence the plea for careful medical study, as has been emphasized by Dr. Tucker and Dr. Patterson.

I was delighted to hear Dr. Tucker refer to careful study of the larynx if we are to make accurate diagnoses. It is well known that some individuals tolerate laryngeal examination to a surprising degree. Then and only then are we able to make out important details as to the extent and distribution of such a lesion, its surface appearance, especially with regard to infiltration, submucous hemorrhage, etc.

Another very important reason for stressing the symptoms of benign lesions of the larynx is the close resemblance of such lesions to those of cancer of the larynx. This was emphasized all the more this morning, when Dr. Jackson made a statement, with which most of us agree, that 90 per cent of the people who have cancer of the larynx die from cancer of the larynx, yet 82 per cent of cordal cancer is curable if seen and treated at the time it should be seen and properly treated. This is all the more reason for a satisfactory laryngological examination of people with unexplained hoarseness.

Of course, the integrity of the cord must be preserved in any operative procedure where the lesion is simple and benign. It is at times impossible to decide the nature of a laryngeal lesion without careful general study as well as biopsy.

When taking a biopsy specimen care must be exercised to cut a section from the surface to the base, inclusive. We have all seen the tragedy in which the patient has been lulled into a false sense of security by having a biopsy taken which was superficial and which included only the inflammatory portion, which many tumors do have; the report was returned as benign, and six months or a year later returned with an inoperable carcinoma of the larynx. Occasionally carcinoma of the vocal cord may have been the site of a submucous hemorrhage, yet when a well secured section is carefully studied with the microscope the base may be found to be a carcinoma.

Voice rest is one of the most important means of treating cases of benign lesions of the larynx, I am sure, and also one of the most difficult to obtain by reason of the fact that so many of these individuals have to make their living by the use of their voices; hence, their lesions and the difficulty in carrying on.

I agree with Dr. Tucker on forbidding the use of strong chemicals in the larynx. I am sure all of us have seen many cases of laryngeal diseases which have been overtreated. The follow-up study of these cases is most important and essential, I think.

The possibility of stimulation of growth of cancer, or of stimulation of transition from precancerous to cancerous disease by biopsy or surgical procedure is one in which I take no stock. We have all seen cancer of the vocal cord remain inactive for a long period and suddenly take on a rapid growth. The diagnosis can be established surely by microscopic study, and should always be carried out.

If all patients with hoarseness could be examined according to the plans suggested by Dr. Tucker, many tragedies would be averted and the death rate for laryngeal disease would be much lower than it is at present.

Instructional Subjects

The American Academy
of Ophthalmology and Otolaryngology

Fortieth Annual Meeting
Cincinnati, 1935

Instructional Subjects

DR. HARRY S. GRADLE, Chicago
Secretary for Ophthalmology, Chairman

DR. RALPH A. FENTON, Portland, Ore.
Temporary Secretary for Otolaryngology

DR. A. D. RUEDEMANN, Cleveland
Associate Secretary for Ophthalmology

DR. DEAN M. LIERLE, Iowa City
Associate Secretary for Otolaryngology

DR. DELBERT K. JUDD, Omaha
Associate Secretary for Mechanical Arrangements

PATHOLOGICAL COURSES

Tuesday Afternoon, Wednesday Morning, Thursday Afternoon

HISTOPATHOLOGY OF THE EYE

DR. MARY KNIGHT ASBURY, Cincinnati
DR. R. C. GAMBLE, Chicago
DR. THEODORE L. TERRY, Boston
DR. GEORGIANA DVORAK THEOBALD, Chicago

HISTOPATHOLOGY OF THE EAR, NOSE AND THROAT

Monday Afternoon, Tuesday Morning, Wednesday Afternoon

DR. JOSEPH C. BECK, Chicago
DR. LEROY A. SCHALL, Boston
DR. GREGOR MCGREGOR, Toronto

CONFERENCES

Tuesday, Wednesday and Thursday Mornings

First Period, 9:00 to 10:30; Second Period, 11:00 to 12:30

* * *

OPHTHALMOLOGY

DR. FRANCIS HEED ADLER, Philadelphia, Pa.
Tuberculosis of the Uveal Tract

DR. THOMAS D. ALLEN, Chicago, Ill.
Systemic Therapy of Ocular Disease — Foreign Proteins, Tuberculins, etc.

DR. CHARLES A. BAHN, New Orleans, La.
Eyes and Endocrines

DR. HANS BARKAN, San Francisco, Calif.
Treatment of Strabismus and Heterophoria by the O'Connor Cinch Operation

DR. OSCAR V. BATSON, Philadelphia, Pa.
The Venous Circulation of the Head

DR. CONRAD BERENS, New York, N. Y.
The Presbyopic Patient with Special Reference to Accommodation, Heterophoria, Refraction and Tension

DR. NELSON MILES BLACK, Miami, Florida
Compensation in Industrial Ophthalmology

DR. ALBERT L. BROWN and
DR. RICHARD S. AUSTIN, Cincinnati, Ohio
Relationship of General Pathology to Eye Pathology

DR. FRANK E. BURCH, St. Paul, Minn.
Complications in Cataract Surgery and Their Management

DR. VIRGIL G. CASTEN, Boston, Mass.
Visual Disturbances in Neurological Conditions

DR. RAMON CASTROVIEJO, New York, N. Y.
Modern Aspects of Corneal Transplantation

DR. MARTIN COHEN, New York, N. Y.
The Measurement and Evaluation of Intraocular Pressure Changes

DR. ALFRED COWAN, Philadelphia, Pa.
Principles and Practice of Retinoscopy

DR. WILLIAM H. CRISP, Denver, Colo.
The Refinements of Refraction

DR. J. H. DUNNINGTON, New York, N. Y.
The Surgical Treatment of Strabismus

DR. LOUISE FARWELL, Evanston, Ill.
Diagnostic Techniques and Treatment of Children with Reading Disabilities

DR. F. BRUCE FRALICK, Ann Arbor, Mich.
Surgical Treatment of Retinal Detachment

DR. ALBERT D. FROST, Columbus, Ohio
Diseases of the Optic Nerve

DR. D. F. GILLETTE, Syracuse, N. Y.
Vitreous Opacities — Etiology, Diagnosis and Treatment

DR. ALLEN GREENWOOD, Boston, Mass.
Practical Ophthalmology. Practical Hints in Practice Gained from Experience

DR. GEORGE P. GUIBOR, Chicago, Ill.
The Orthoptic Treatment of Squint in Children

DR. PARKER HEATH, Detroit, Mich.
Ophthalmic Therapeutics

DR. SEARLE B. MARLOW, Syracuse, N. Y.
The Routine Testing for, and Management of, Heterophoria

DR. LUTHER C. PETER, Philadelphia, Pa.
The Diagnostic Value of Perimetry

DR. A. B. REESE, New York, N. Y.
The Operative Treatment of Glaucoma

DR. BERNARD SAMUELS and
MR. EDGAR B. BURCHELL, New York, N. Y.
The Morbid Anatomy of the Retina

DR. MARK J. SCHOENBERG, New York, N. Y.
Acute Glaucoma

DR. PHILLIPS THYGESON, Iowa City, Iowa
Bacteriology of the Conjunctiva — Theory and Practice

DR. ROBERT VON DER HEYDT, Chicago, Ill.
Clinical Photography of the Eye

DR. HENRY P. WAGENER, Rochester, Minn.
Retinitis of Hypertension and Arteriosclerosis

DR. JOHN M. WHEELER, New York, N. Y.
Plastic Surgery of the Eye Region

OTOLARYNGOLOGY

DR. JOHN F. BARNHILL, Miami Beach, Fla.
The Larynx — Anatomy and Associated Phenomena

DR. OSCAR V. BATSON, Philadelphia, Pa.
Demonstration of the Surgical Approaches to the Neck

DR. OSCAR V. BATSON, Philadelphia, Pa.
The Venous Circulation of the Head

DR. HARRY P. CAHILL, Boston, Mass.
The Diagnosis of Intracranial Lesions

DR. CLAUDE C. CODY, JR., Houston, Texas
Deficiency Diseases in Otolaryngology

DR. CHARLES E. CONNOR, St. Paul, Minn.
Laboratory Aids to Diagnosis in Otolaryngology

DR. WARREN B. DAVIS, Philadelphia, Pa.
The Development and Anatomy of the Paranasal Sinuses

DR. LEE W. DEAN, St. Louis, Mo.
Pediatric Otolaryngology

DR. HARRY N. GLICK, St. Louis, Mo.
Petrous Apex Infection

DR. PERRY G. GOLDSMITH, Toronto, Canada
Failures and Errors in the Practice of Otolaryngology

DR. W. E. GROVE, Milwaukee, Wis.
The Ear and Head Injuries

DR. FREDERICK T. HILL, Waterville, Maine
Acute Throat Conditions

DR. CHEVALIER JACKSON, Philadelphia, Pa.
Hoarseness

DR. MARVIN F. JONES, New York, N. Y.
Labyrinthitis

DR. SAMUEL KOPETZKY, New York, N. Y.
The Radical Mastoid Operation

DR. FRANCIS L. LEDERER, Chicago, Ill.
Cholesteatoma

DR. GEORGE S. LIVINGSTON, Chicago, Ill.
The Management of Sinus Diseases in Children

DR. WILLIAM J. McNALLY, Montreal, Canada
Anatomy and Physiologic Phenomena of the Internal Ear

DR. PHILIP E. MELTZER, Boston, Mass.
The Mastoid Cells — Their Arrangement in Relation to the Sigmoid Portion of the Transverse Sinus

DR. WILLIAM MITHOEFER, Cincinnati, Ohio
Therapy of Nasal Accessory Sinus Diseases

DR. HARRIS P. MOSHER, Boston, Mass.
Anatomy and Pathology of the Œsophagus

DR. JOHN L. MYERS, Kansas City, Mo.
Physical Therapy in Otolaryngology

DR. GORDON B. NEW, Rochester, Minn.
Malignancies of the Upper Respiratory Tract

DR. A. G. POHLMAN, Omaha, Nebr.
Auditory Test Methods from the Viewpoint of Practicability in Diagnosis

DR. ROBERT F. RIDPATH, Philadelphia, Pa.
The Sphenoid Sinus

DR. EDWARD C. SEWELL, San Francisco, Calif.
Inflammatory Diseases of Nose and Sinuses

DR. BENJAMIN H. SHUSTER, Philadelphia, Pa.
Tinnitus Aurium and Vertigo

DR. JOHN R. SIMPSON, Pittsburgh, Pa.
Minor Conditions Involving the Ear, Nose and Throat

Presentation of Instruments

The American Academy
of Ophthalmology and Otolaryngology

* * *

Fortieth Annual Meeting
Cincinnati • 1935

A COMBINATION LOUPE AND HEAD MIRROR

CONRAD BERENS, M.D.

NEW YORK

BECAUSE it has been found troublesome to remove the loupe attached to my spectacles in order to use a head mirror a combination loupe and head mirror* (Fig. 1) was devised.

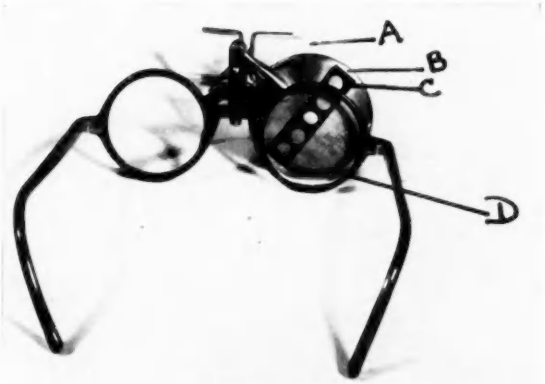


FIG. 1 — (Berens) — Head mirror with loupe and attachments for spectacle frame. *A*, modified Beebe loupe. *B*, head mirror. *C*, bar for insertion of plus lenses. *D*, bifocal segment.

The description of the loupe attached to the spectacle frame, both with and without a light, was previously published.[1] The addition of the concave head mirror, 55 mm. in diameter with an aperture 6 mm. in diameter, seems to be practical not only for physicians treating diseases of the ear, nose and throat, but it is useful also for the ophthalmologist who wishes to free both hands when examining and treating his patients. A sliding bar with an aperture and four cells to hold lenses adds to the value of the mirror for the presbyopic surgeon. The preferred lenses are +1.00, +2.00, +3.00, and +4.00.

* Made by the E. B. Meyrowitz Company, New York, N. Y.

1. Berens, C., and Losey, R. R.: *A New Arrangement of the Beebe Loupe*, Tr. Sect. Ophth., A.M.A., 1928, p. 417.

A SIX-METER RECORDING STEREOSCOPE

CONRAD BERENS, M.D.

NEW YORK

THE IMPORTANCE of the time factor in the study of stereopsis was the principal reason for devising a six-meter recording stereoscope.

This stereoscope (Fig. 1) automatically controls the time factor, as the speed of movement of one milk glass plate can be changed by moving lever E and the rapidity with which the tests

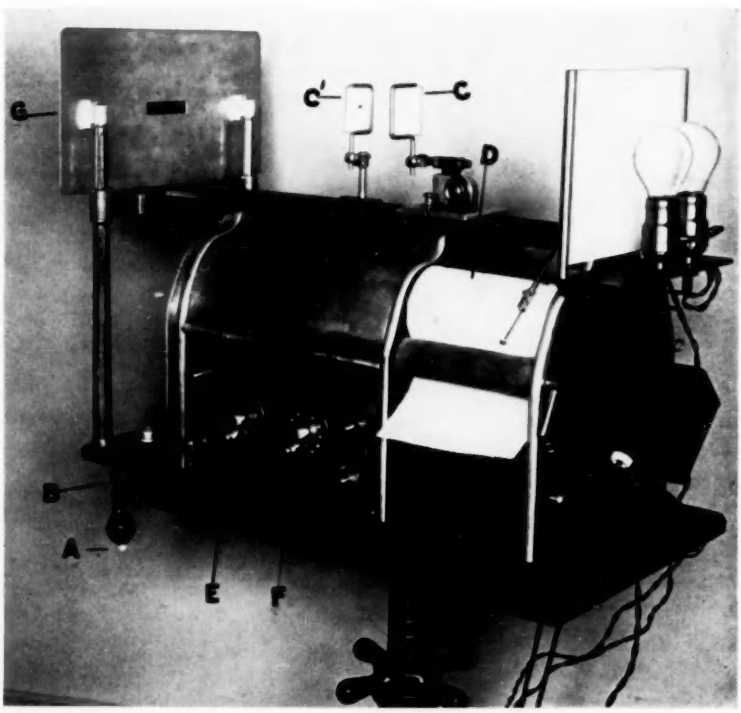


FIG. 1 — (Berens) — A six-meter recording stereoscope which automatically controls the time factor. *A*, push button. *B*, six meter cord. *C* and *C'*, milk-glass plates as test objects. *D*, continuous roll of paper for recording judgments. *E*, lever for regulating speed of carrier (*C*). *F*, lever for adjusting the length of the rest period. *G*, metal screen with slit through which milk-glass plates are viewed.

are made by adjusting lever F. A push button (A) attached to a six-meter cord (B) permits the instrument to be used at any desired distance. Milk-glass plates (C) are used instead of rods.

Records are made with a continuous roll of paper (D). The record of a subject who had excellent stereoscopic ability may be seen by referring to figure 2. The single horizontal line indicates the position of the stationary test object and shows the accuracy with which the subject made his judgments. This stationary rod may be moved and locked in a new position so that accuracy may be checked.

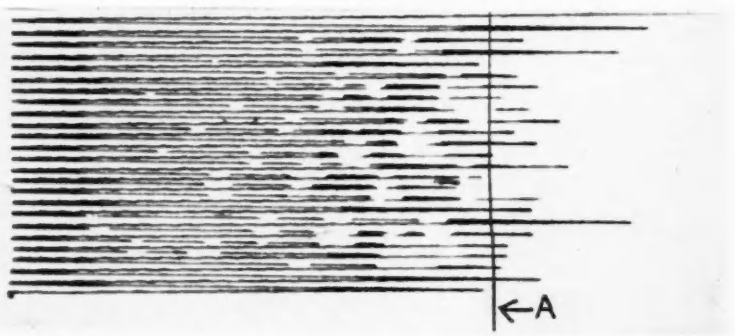


FIG. 2 — (Berens) — Record of stereopsis of a subject who had had excellent training in studying aerial maps with a stereoscope. Line *A* indicates the position of the stationary test object (milk-glass plate). Record made with the six-meter recording stereoscope.

A STRABISMOMETER

J. L. Bressler, M.D.

CHICAGO

Some of the advantages of this strabismometer are the following:

1. The instrument slides up and down in a tubular stand. It can be firmly fixed at any desired height to accommodate varying heights of patients.

2. It cannot sway from side to side, nor up and down. Once centered and fixed, it remains so. The eyepiece, which is 33 cm. from the center of the arc, is so marked that accurate centration of the cornea is neither difficult nor a matter of guesswork.

3. The arc consists of a steel bar, about 1 cm. in width, over which the patient can readily fix the distant light, and over which the examiner can readily see the reflex of his ophthalmoscopic light on the patient's cornea.

4. The arc revolves about a swivel joint and can be placed in any meridian. This facilitates the measuring of angles in any position. It also makes possible the measuring of the limits of motility of each eye in any meridian.

5. The arc is calibrated in single degrees.

6. For the near fixation point there is a movable target, which can be placed in position when needed.

7. The time consumed in measuring an angle with this instrument, both for distance and near, averages from one and one-half to two minutes. In very young children it is just as useful as in older children.

8. This instrument can be so built that it may be used as an ordinary perimeter when the eyepiece is removed.

* * *

I am indebted to Mr. Freeman of the American Optical Company in Chicago for his help in making possible the building of this instrument.

Strabismometer (Bressler). *A*, Eyepiece. *B*, Arc. *C*, Chin rest. *D*, Movable target. *E*, Sliding rod. *F*, Stand.

AN ILLUMINATING DEVICE TO BE USED AS AN ATTACHMENT TO THE BINOCULAR CORNEAL MICROSCOPE FOR GONIOSCOPY AND GONIOPHOTOGRAPHY

RAMON CASTROVIEJO, M.D.

NEW YORK

THE OBJECT of this presentation is to demonstrate an illuminating device that can be attached to any standard corneal microscope, which, thus equipped, may be used for gonioscopy and goniophotography.* With the aid of the contact glass for fundus examination, and the corneal microscope equipped with the illuminating device, observations of the fundus can be made with high magnifications.

The instrument (Fig. 1) consists of a 3.5 V. 3.8 Amps. straight filament lamp of special design, adjustably mounted in a vertical lamp housing (Fig. 1-a). Below the lamp are mounted a totally reflecting prism of 87° deviation and a condenser of 2.5 cm. focal length (Fig. 1-b). The vertical adjustment of the filament near the focal plane of the condenser permits the use of a collimated, diverging, or converging beam, as desired, of narrow cross-section and high intensity. The illuminating system, as a unit, is mounted on a tilting hinge for adjustment (Fig. 1-c) at various angles to the axis of the microscope, to avoid reflexes. Back of the hinge joint, a vertical adjustment by a quick-acting screw (Fig. 1-d) permits a rapid leveling of the entire accessory.

This device attaches directly to the mounting of the binocular corneal microscope by means of a clamp screw (Fig. 1-e, and Fig. 2). Located between the objective mounts, it provides central illumination close to the object, with provision made on the mounting to avoid direct reflex into the objectives of the microscope. The axis of illumination in relation to the axis of the objectives may vary vertically between 3° and 18°.

* Castroviejo, Ramon: *Goniophotography*, American Journal of Ophthalmology, Vol. 18, No. 6, (June) 1935.

GONIOSCOPY AND GONIOPHOTOGRAPHY IN HUMAN EYES

The patient is placed in the recumbent position and the eye anesthetized with several instillations of holocaine hydrochloride, 1% solution. The conjunctival sac is freely irrigated with normal saline solution to wash out mucus and other foreign substances which, interposed between the contact glass and the cornea, may disturb the observation. The Zeiss-Koeppe contact glass with full hemisphere for iridic angle, also called "C" glass when made by Bausch and Lomb Optical Company of Rochester,

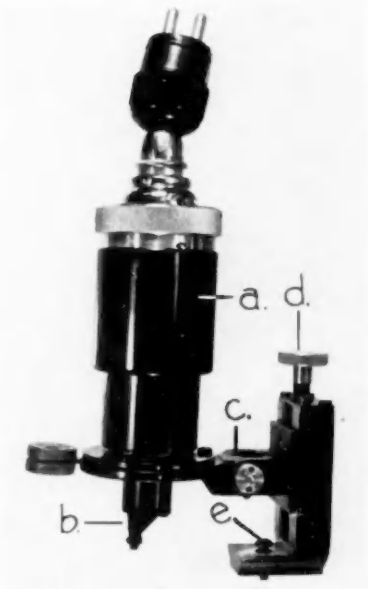


FIG. 1 — (Castroviejo) — Author's illuminating device.

FIG. 2 — (Castroviejo) — Illuminating device mounted on the binocular corneal microscope.

N. Y., is placed in position with the margins of the eyelids surrounding the glass and lying in the groove around its border. The glass is thus held by the eyelids and pressed tightly against the eyeball. Normal saline is then injected to fill the space left between the concave surface of the contact glass and the cornea. For this maneuver, a small canula attached to a rubber bulb, similar to that which is used for the irrigation of the anterior chamber of the eye, may be employed. The external surface of the glass is then washed with distilled water and carefully dried

with a piece of gauze. If the saline solution comes in contact with the external surface of the glass, the rapid evaporation of the fluid leaves a coating of salt deposit which greatly disturbs the view.

The observation of the angle is made with the microscope, equipped with the illuminating device, as indicated in Fig. 3. Different magnifications can be obtained by using different objectives, but best results will be obtained with the objectives of low power, which, having more depth of focus, permit the simultaneous observation of different planes. The stereoscopic view of the angle of the anterior chamber obtained with the binocular microscope, used as described, provides greater ease in the interpretation of histological and pathological details which may be puzzling when monocular observations are employed. For demonstration purposes the binocular microscope may be used by two observers simultaneously, each one looking through a different ocular. In this fashion the instructor points out the interesting details to be observed by the second person and thus it becomes most useful for teaching.

If a photograph of the angle is desired, one of the oculars is replaced by a miniature camera (Fig. 4), equipped with a lateral view finder. The observation of the angle is made through the lateral view finder. When the desired field of the angle has been brought into focus, the prism action which deviates the image towards the lateral view finder is automatically interrupted by making exposure with a special cable release. Photographs may be taken with exposure of $\frac{1}{20}$ of a second (Fig. 5).

For photographic purposes the microscope with the accessories should be mounted upon a heavy compound base for the binocular microscope, which permits a free and fully controlled movement of the camera in every desired direction for adjustment and focusing without being subject to vibrations.

EXAMINATION OF THE FUNDUS

For this purpose the most recent Zeiss model of contact glass for fundus examination is required. This contact glass has a flat anterior surface of 15 mm. diameter, the previous model having a 9 mm. flat surface. The new model makes it easier for the pupil to lie beneath the examining area of the glass, when it is placed in position. The pupil should be widely

FIG. 3. — (Castroviejo) — Photograph showing how the examination of the angle of the anterior chamber is conducted.

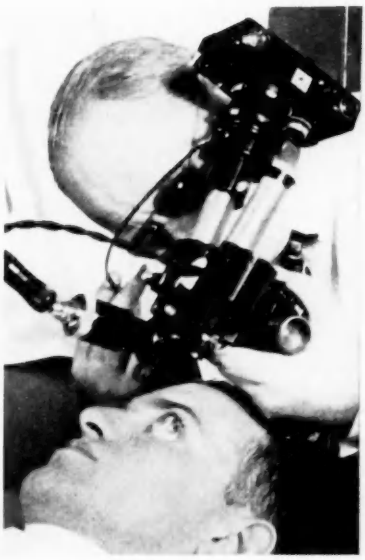


FIG. 4. — (Castroviejo) — Photograph showing how the equipment is assembled for photography of the angle.

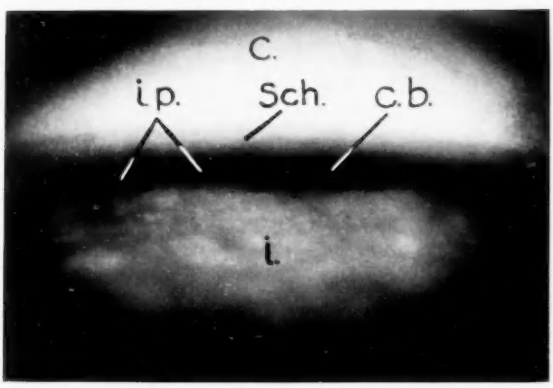


FIG. 5. — (Castroviejo) — Photograph of the angle of the anterior chamber of a normal eye. *C*, Cornea. *Sch.*, Schlemm's canal. *C.b.*, Ciliary body. *I.p.*, Iris processes. *I*, Iris.

dilated, the contact glass placed in position and the space between the eye and the glass filled with normal saline solution. The observation is now made in the same way as when the anterior segment of the eye is examined with the binocular microscope and the slit-lamp. The patient sitting down, the head kept steady in the head and chin rest, and the fundus is observed with the binocular microscope equipped with the illuminating device. It is convenient to have the microscope mounted on a heavy stand, equipped with a cross slide adjustable movement, to facilitate focusing.

Very clear observations of the fundus can be made in this way with magnifications ranging from 15 to 60 diameters, depending on the objectives used. The author has been able to obtain photographs of the fundus, using the equipment described above, but for this it is necessary to have a maximum dilatation of the pupil and a very cooperative patient. However, photography of the fundus with standard cameras and methods which do not necessitate the use of contact glasses is more practical. I wish to express my appreciation to Mr. Victor M. E. Koch, of Carl Zeiss, Inc. — New York Agents for Carl Zeiss, Jena — for his cooperation in making the instrument here presented.

A NEW METHOD FOR DETERMINING THE BLURREDNESS OF THE RETINAL IMAGE

GLENN A. FRY, Ph.D.,[1] AND
PERCY W. COBB, M.D.

ST. LOUIS

OBVIOUSLY the most accurate method for determining the blurredness of the retinal image is to calculate the distribution of light in the image of a point source of light in accordance with the laws of chromatic and spherical aberration, and diffraction, and once this is determined the distribution of light in any stimulus pattern can be calculated.

This method is tedious, however, and does not take into account the fine rapid oscillations of the eye which, in effect, constitute another source of blurring.[2] We have advised, therefore, another method which is simple and gives an overall measure of the total degree of blurring produced from the various sources. The method rests upon the assumption that the distribution of light across the image of an indefinitely narrow bar follows the Gaussian equation

$$[1] \qquad Y = \frac{1}{\sigma\sqrt{2\pi}}\, e^{-\frac{X^2}{2\sigma^2}}$$

in which Y is the intensity of the image at a distance X from the mean. The equation is here written so that σ is the standard deviation of the mean square deviation from the mean of the distribution, and so that the area under the curve between plus and minus infinity is equal to unity. How much the actual distribution of light in the image departs from this equation is not known, but any distribution which approximates this form will

1. The leading author's part in this investigation was made possible by a grant from the American Academy of Ophthalmology and Otolaryngology.

2. Adler, H. F., and Fliegelman, M.: *Influence of Fixation Upon Visual Acuity*, Arch. Ophth., Chicago, 12:475-83, 1934.

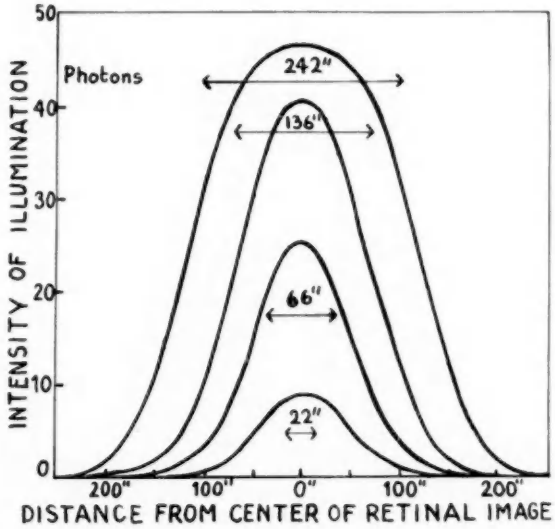


FIG. 1 — (Fry and Cobb) — Distribution of light across the retinal images of bars of different width, when the brightness of the bars is 1 c. per sq. ft. and the diameter of the artificial pupil 2.33 mm. It is assumed that the light in the retinal image of an infinitely narrow bar is distributed according to the Gaussian equation with σ equal to 44" of visual angle.

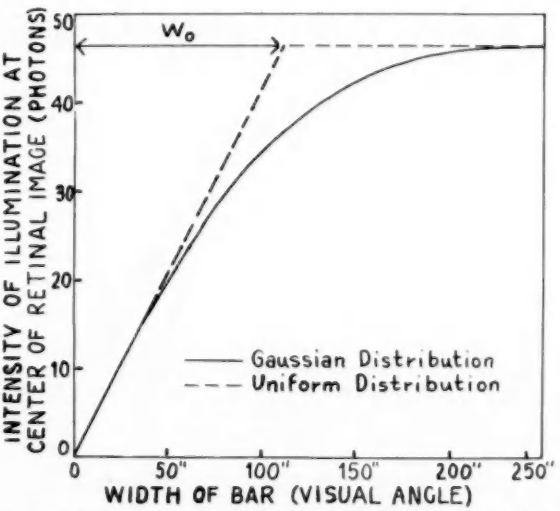


FIG. 2 — (Fry and Cobb) — The relation between the width of a bar and the intensity of illumination at the center of the retinal image. One curve is based upon the assumption that the light in the image of an infinitely narrow bar is distributed according to the Gaussian equation with σ equal to 44" of visual angle. The other curve assumes that the light is distributed uniformly across a strip 110" wide.

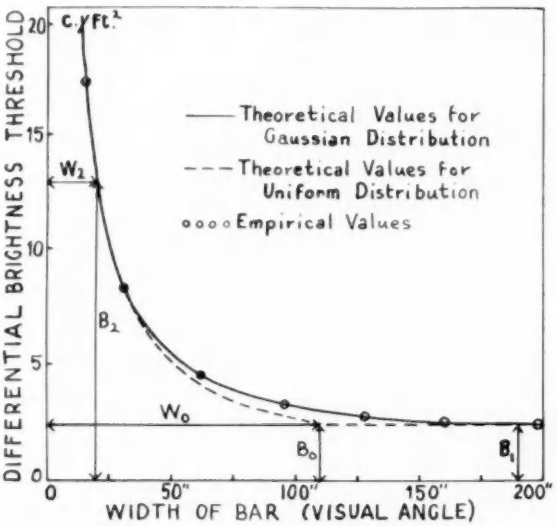


FIG. 3 — (Fry and Cobb) — The effect of varying the width of a bar upon its brightness difference threshold. One set of theoretical values is based upon the assumption that the light in the retinal image of an infinitely narrow bar is distributed according to the Gaussian equation with σ equal to 44″ of visual angle. The other set of theoretical values is based upon the assumption that the light is distributed uniformly across a strip 110″ wide. The empirical values were obtained with a bar 3000″ long on a large disk-shaped field, subtending a visual angle of 2° 46′, and having a brightness of 50 c. per sq. ft. Each value represents an average of ten readings.

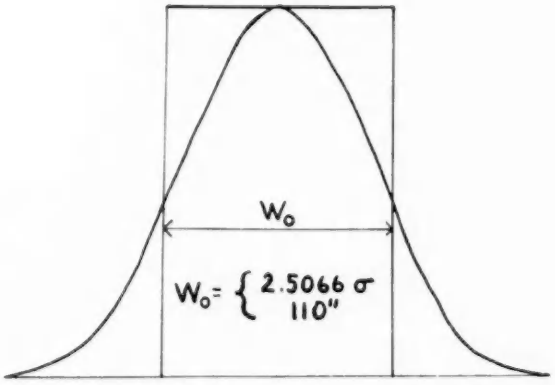


FIG. 4 — (Fry and Cobb) — Gaussian and uniform distributions of light in the retinal image of an infinitely narrow bar. Since the total amount of light is the same for the two cases, W_0 is equal to 2.5066 σ.

yield essentially the same results. When this assumption is made the problem of measuring the blur consists simply in measuring the value of σ.

For the sake of clearness, we shall not start with empirical data and calculate backward to obtain σ, but we shall assume that σ is equal to 44″ of visual angle and show that this value checks with experimental facts. Our method for determining σ is based upon the fact that the threshold of visibility of a narrow bar depends upon the width as well as the brightness of the stimulus. We have assumed that this relation depends, in turn, upon the fact that the threshold of visibility is determined by a certain minimum intensity at the center or maximum of the retinal image, and that the effect of reducing the width of the bar is to lower the intensity at the center (owing to the blur), and that the change in brightness necessary to bring the bar back to threshold merely compensates the loss in intensity so incurred by reducing the width.

The change in intensity at the center of the retinal image with change in the width of the bar is determined by the blurredness of the image. If it is assumed that the light in the retinal image of an infinitely narrow bar is distributed according to the Gaussian equation with σ equal to 44″, it is possible to calculate the distribution for wider bars and to show how the width of the bar affects the intensity at the center of the image.[3] Distributions for various widths of bar are shown in Fig. 1, and the unbroken curve in Fig. 2 shows the relation between the width of a bar and the intensity of illumination at the center of the retinal image. The intensity increases as the width increases at first nearly proportionately, then more slowly, and beyond 242″ further increase in intensity is negligible. The intensity of retinal illumination for any bar wider than this is equal to 1 photon per sq. mm. of pupillary area and per c. per sq. m. of the external surface. This, as a matter of fact, is the definition of a photon. A pupil 2.33 mm. in diameter, which we have used in our experiments, gives 46.5 photons per c. per sq. ft.

If one assumes that at the threshold, the intensity of the center of the retinal image remains constant regardless of width, and if one knows the threshold stimulus brightness for a bar of any given width, he should be able to calculate the threshold

3. The calculation of these various distributions was actually made with the help of the table of integrated values of equation (1) for the various values of X, in H. E. Garrett, *Statistics in Education and Psychology*, p. 91, New York, 1926. Similar tables are to be found elsewhere.

brightness for a bar of any other width from the data in Fig. 2, because the threshold brightness for any width is equal to the threshold intensity at the center of the retinal image divided by the number of photons per unit brightness which the width in question is capable of producing.[4]

We have in this way calculated the theoretical values for the brightness difference threshold of a bar, 3000″ long, upon a slightly darker field which was disk shaped, subtended a visual angle of 2° 46′, and possessed a brightness of 50 c. per sq. ft. First of all we measured the empirical threshold for a width of 192″ and then calculated the theoretical values for the other widths, which are represented by the unbroken curve in Fig. 3. Empirical values have also been obtained and are represented by the circles. The fact that the empirical and theoretical values coincide validates our original assumptions, (1) that the threshold intensity at the center of the retinal image remains constant for different widths, (2) that the distribution of light in the retinal image of an infinitely narrow bar approximates the Gaussian equation, and (3) that σ is equal to 44″.

This value for σ need not be expected to hold for other conditions than we have used, namely, white light, an artificial pupil 2.33 mm. in diameter, and a well accommodated eye. The use of monochromatic light would do away with the effects of chromatic aberration and thus reduce the blur. If the pupil were made smaller the effects of diffraction would be increased and the effects of chromatic aberration decreased, and *vice versa*, if the pupil were made larger.

In practice it is necessary to start with the empirical data and calculate backward to σ. In order to determine σ it is only necessary to measure the brightness threshold (B_1 and B_2) for bars of two widths, one (W_1) so wide that further increase in width will not lower the threshold, and one (W_2) so narrow that a reciprocal relation holds between brightness and width at threshold. Any width of more than 242″ will suffice for W_1 and any width narrower than 30″ for W_2. σ can then be calculated according the following formula:

$$[2] \qquad \sigma = \frac{1}{2.5066}\left(\frac{B_2 W_2}{B_1}\right)$$

4. It is to be noted that the term "brightness" refers to the external stimulus, whereas "intensity" applies to the illumination in the blurred retinal image on the retina.

The derivation of this formula is as follows: In Fig. 2 it can be seen that for narrow bars, the intensity at the center of the image is proportional to the width of the bar. If the distribution of light in the retinal image of an infinitely narrow bar were uniform instead of bell-shaped, as shown in Fig. 4, the proportionality between intensity and width would continue to hold until the width of the bar reached the width of the rectangle in Fig. 4, but after this the intensity would remain the same. This maximum intensity must be the same for the rectangle as for the Gaussian distribution when the area of the rectangle is equal to that under the curve. This is true when

$$[3] \qquad \frac{\frac{1}{2}W_0}{\sigma} = \sqrt{\frac{\pi}{2}} \quad , \text{ or } \quad W_0 = 2.5066\sigma$$

If the relation between width and retinal illumination in Fig. 2 is proportional, as indicated by the oblique dotted line, the relation between width and brightness in Fig. 3 must be reciprocal as indicated by the dotted hyperbola. W_0 in this figure is determined by the point where the hyperbola reaches the level of the horizontal limb of the curve. According to the hyperbolic relation

$$[4] \qquad W_0 B_0 = W_2 B_2$$

but $B_0 = B_1$, and $W_0 = 2.5066$, and when these values are substituted in Formula 4, Formula 2 is obtained.

Summary — A method has been described for determining the degree of blurredness of the retinal image which is based upon the assumptions (1) that the light in the retinal image of an infinitely narrow bar is distributed according to the Gaussian equation, and (2) that the dependence of the threshold of visibility of a narrow bar upon its width is an outcome of the fact that the retinal image is blurred and that changing the width of the bar changes the intensity at the center of the retinal image and (3) that the threshold of visibility is determined by a certain minimum intensity at the center of the retinal image. These assumptions are validated by experimental facts.

An extension of this type of work to the discrimination of two parallel bars has been completed and is at present in publication.[5]

5. Fry, G. A., and Cobb, P. W.: *The Visual Discrimination of Two Parallel Bright Bars in a Dark Field*, (now in press).

SCIENTIFIC AND PRACTICAL CONSIDERATIONS INVOLVED IN THE NEAR VISION TEST WITH PRESENTATION OF A PRACTICAL AND INFORMATIVE NEAR VISION CHART

JAMES E. LEBENSOHN, M.D., Ph.D.*

CHICAGO

THOUGH near vision charts were designed before those for distant vision, no model has received an acceptance comparable to the Snellen distance chart, since none has satisfactorily surmounted the many special difficulties inherent in near vision testing.

RETROSPECT

The idea of arraying test-letters in series originated with Küchler, who in 1836 presented a near vision test with a letter gradation from 1.5 mm. (9 point) to 21.5 mm. (120 point). Jaeger in 1857 published in Vienna an extended series, arbitrarily numbered from 1 to 20, and ranging from 0.5 mm. (3 point) to 19.5 mm. (110 point). The type was never standardized and the various editions show considerable divergency in the size, shape, and character of the fonts used.

At Snellen's suggestion (1862), reading charts have since been constructed in which the height of the small lower case letters subtends a five minute visual angle at stated distances of 0.37 m., 0.50 m., etc. For the abbreviation of meter, there has been unfortunately substituted in many cards the letter D of the Snellen formula, $V = d/D$. Since "D" is the abbreviation of diopter, a curious and fairly widespread confusion has resulted in many men referring to such charts as in the "diopter" system. On one of these charts the refractionist is instructed that "The number over the paragraph will be about the number of convex glasses required for reading and sewing." Of course, such a correlation, though it does occur above 1.00 diopter, is purely accidental.

* From the Department of Ophthalmology, Northwestern University Medical School, Chicago, Ill.

However, it is impossible to present reading matter in lower case that precisely conforms to the mathematical requirements of physiologic optics. Consequently, in 1885 Oliver[1] advocated that short words in capitals be used instead. From the practical standpoint the unrealistic nature of such a test is objectionable; and the psychologist would not consider that words and the optotypes from which they were formed are on the same level of recognition. The recent A.M.A. card for rating visual efficiency at the near point is based on the Oliver chart, but has been modified for use at fourteen inches, and does not confine itself to the seven letters, C D E T O L F, which Oliver considered alone suitable.

In correcting the presbyopia of illiterates the ancient Chinese had the individual examined look at the ridges of his finger-tips. The limited intelligence of the illiterate requires a test of such simplicity. The geometrical symbols devised by Burchardt (1870), Landolt (1888), Fridenberg (1910),[2] Dor (1920),[3] and the pictographs of Ewing (1920),[4] though simple, are not simple enough; and moreover fail to arouse interest, even in the adult illiterate.

REQUIREMENTS

The essential weakness of our present reading charts lies in their over-simplification. A single chart attempts to cover the diversified scientific and practical demands of near vision testing, whereas two distinctly different charts are requisite: (1) simple optotypes based on the one minute visual angle as in the distance charts; (2) a standardized reading text in the various sizes of customary print.

In addition the doctor would do well to gather in a scrap book empiric tests adapted to the individual needs of his clientele, such as cloth with needle and thread; clippings from newspapers and magazines; excerpts from the telephone directory, want-ads, and market reports; specimens of music, playing-cards, road maps, and blue-prints; samples of handwriting, typewriting, and shorthand; and, when needed, selections from foreign languages.

MINIATURE OPTOTYPES

The reduced optotype is the only scientifically accurate means by which the visual acuity for near can be ascertained. Reading texts can only approximately fulfill the same task. The selected distance chart can be reduced by photographic methods to give

equivalent values at whatever near distance is decided upon as standard (Bjerke[5]). With a reduction to 1/17, the six meter line subtends a five minute angle at nearly fourteen inches (exactly 13.9 inches, 35.3 cm.). This appears the most advantageous reduction as it is in harmony with the efforts of the American Medical Association to standardize near vision tests at fourteen inches. With the miniature charts, as in distance testing, both a letter and number series are desirable.

CORRELATION OF NEAR AND DISTANCE VISION

The visual acuity of the normal accommodating eye, taken with the vertex of the correcting lens at the anterior focal point, is the same for distance and near. This is the result obtained by Bjerke[6] and myself employing reduced Snellen charts, by Landolt[7] using a miniature broken-ring chart, and by Snell[8] with the Oliver chart. Hegner[9] finds from theoretical considerations also that the effect of accommodation should be negligible.

Biedermann,[10] however, reported an increased visual acuity for near, but he made his tests at 25 cm. (9.8 in.) with a miniature Landolt chart produced on a milk-white transparency; and Brückner[11] would discount his findings as probably due to small pupil, variations in illumination and attention, etc. Though it is true that objects subtending the same visual angle do not appear the same size at different distances (Aubert-Förster phenomenon), this is a purely psychological interpretation, and certainly does not affect the discriminatory power of the retina.

In presbyopia, the correcting lens not only restores the visual acuity for near, but increases the visual image 1.5 per cent for each diopter added (Bjerke[12]). In aphakia, the anterior focal point is two centimeters or more from the corneal apex, and a correcting lens placed there would produce an image 1.5 times normal size. But as the lens is generally one centimeter nearer to the eye, this possible acuity is reduced 1 per cent for each diopter (Bjerke[12]).

In abnormal eyes, Snell[8] notes that the visual acuity for distance and near is generally parallel, excepting where the cornea or lens is involved, when there may be a significant difference.

READING TESTS

"When the patient reading Jaeger 1 or Thorington 0.50 D asks how this compares with the newspaper, one is not able to answer definitely, as the relative value is not apparent." (Wells[13]).

Reading is the predominant interest in near vision, yet (because of considerations which are better realized in reduced optotypes) the reading tests in use have no practical relation to reading experience. From this standpoint an arrangement of a standardized type in the point system would be definitely more useful.

As is now well known, we read in thought units and recognize words largely by their shape.[14,15] Because of such associations, words may be read when the component letters are so small that they could not be individually identified. Much of the characteristic appearance of a word is due to the fact that there are three forms of lower case letters: ascenders, descenders, and small sorts. Hence, to avoid the pitfalls of context and configuration, I would suggest that for the continuous text in our charts there be substituted a series of unconnected words composed wholly of small lower case letters. This presents no difficulty, since nearly all the vowels and most of the more frequently occurring consonants are in this class. The idea is applicable to all languages using Latin letters, thus:

ENGLISH: arc room mason omen crane verse scan cocoa cornea acne
FRENCH: accuser amas amazone amoureux ananas assurance ancon
SPANISH: acaecer acaro amarra amor amansor accesso azuzar ave
ITALIAN: acume acro acrezza acre acervo accusare ace accrescenza
GERMAN: anmassen arm azurn man aus namens neu nun rosa sauer
(The above is 8 point Century Expanded)

THE POINT SYSTEM

In the point system the various sizes of type bodies bear a fixed and simple relation to one another. According to the American system — adopted in the United States in 1886, and in England in 1898 — the point is nearly $\frac{1}{72}$ inch or 0.35 mm. The basic idea originated with Fournier in 1737. Benjamin Franklin, while in France, purchased some of Fournier's foundry materials, which in time came into the possession of the leading American typefounding company, and became the models for their later designs. After the Chicago fire, John Marder, whose typefoundry had been destroyed, moved the adoption of a point system based on their pica type, then in most general use. Consequently, the Fournier system, used in Belgium and parts of France and Austria, and the American system are practically identical.

The point system refers to the size of the type body without reference to the print. In any particular type design, however, the heights of the various characters bear a definite relationship to the size of the type. Thus in Century Schoolbook type the small sorts are just half the point size, so that when the body is 10 point, their height is $^{7}/_{72}$ inch or 1.75 mm. (5×0.35 mm.).

CENTURY SCHOOLBOOK TYPE

The Century Schoolbook type seems the most suitable design on which to standardize our reading tests since it is the result of an effort to produce the most legible type possible. The project was inspired by the Reports of the Committee to Inquire into the Influence of Schoolbooks upon Eyesight (1911-1913) appointed by the British Association for the Advancement of Science.

The design of Century Schoolbook is simple, fairly broad, with fairly thick lines, fairly wide spacing, and with not too much contrast of thickness and thinness. It is based upon the popular Century Expanded, an Old-Style face created by De Vinne, the master type-designer of modern times.

PRESENTATION OF AN ORIGINAL CHART

Description — The two cards submitted are intended to supply the examiner conveniently with essential informative data as well as to provide for nearly all the requirements of near vision testing. The first chart contains an explanatory paragraph; optotypes in two series; letters and numbers, and a staff of music. The second chart consists of three columns: words in point type; numbers of corresponding size, and an illiterate test.

The Optotypes — The optotypes on the chart give a sequence of distance equivalent from 20/13 to 20/800. The distance Snellen charts from which the reductions were made were selected because they are among those most widely used, have an adequate range, and a corresponding letter and number series. The lower group (20/13 to 20/100 equivalent) represents a $^{1}/_{17}$ reduction; the upper group (20/200 to 20/800 equivalent) a reduction of $^{4}/_{17}$.

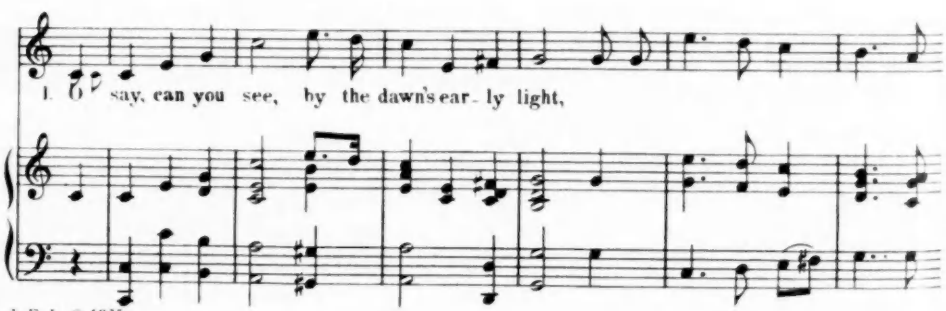
For each line the visual acuity is noted in four sets of interpretative data: decimal notation; reading norm (distance at which letter or number subtends the five minute angle) in centimeters and inches; distance equivalent; and visual efficiency.

NEAR VISION TEST CHARTS

As designed by Dr. James E. Lebensohn,
Northwestern University Medical School, Chicago

The charts are to be read at 35 cm. (14 inches); (exactly 35.3 cm.; 13.9 in.) The reading visual acuity obtained from the miniature Snellen charts can be expressed in terms of distance equivalent, visual efficiency, or—as with corresponding distance charts—decimally, or in fractions. Thus "normal" reading visual acuity 1.0 can be recorded: 20/20 equivalent; 100% efficiency; 35/35; or 14/14;—reading visual acuity 0.8 as : 20/25 equivalent; 95.6% efficiency; 35/44; or 14/17. The adjoining test with point-type (Century Schoolbook) indicates what this means in practical reading ability. The left column therein is for illiterates who are simply to count the "crosses" in each group as in : oxxo oxoxx.

Decimal notation	Reading norm Cm.	Reading norm In.		Distance equivalent	Visual efficiency
0.025	1412	560	L 85	20/800	0.1%
0.050	706	280	F D 293	20/400	5.3%
0.075	471	187	E C T 8754	20/260	11.7%
0.1	353	140	L Z T D 63952	20/200	20.0%
0.2	176	70	F P 293	20/100	48.9%
0.3	118	47	T O Z 8754	20/65	66.8%
0.4	88	35	L P E D 63952	20/50	76.5%
0.5	71	28	P E C F D 428359	20/40	83.6%
0.6	59	23	E D F C Z P 3746285	20/30	91.4%
0.8	44	17	F E L O P E D 9370865	20/25	95.6%
1.0	35	14	D E F P O T E C	20/20	100.0%
1.2	29	12	L E F O D P C T	20/16	
1.5	24	9		20/14	



J. E. L. © 1935

Letter height—4.2164 mm.	*24 Point*	*Acuity: Approx. 0.12 (20/170) Sight-saving texts.*
man oxen	25 37 84 90	OXXO OXXX
Letter height—2.2512 mm.	*18 Point*	*Acuity: Approx. 0.15 (20/130) Books, children 7-8 yrs.*
raw see van	90 89 76 60 54	XOXXX OXOOO
Letter height—2.5146 mm.	*14 Point*	*Acuity: Approx. 0.2 (20/100) Books, children 8-9 yrs.*
noon even mew	38 72 80 93 54 60 76	XOXX XXXX OOOX
Letter height—2.0828 mm.	*12 Point*	*Acuity: Approx. 0.25 (20/80) Books, children 9-12 yrs.*
war use worm eve	40 53 82 64 79 60 47	XOOX OOOXO OOXXX
avenue ransom err	809 423 657 980 765	XOXXO XOXOX OXOOX
Letter height—1.5726 mm.	*10 Point*	*Acuity: Approx. 0.28 (20/70) Adult textbooks.*
scum crease nervous	98 67 45 34 23 50 86 73 98	XOOO OXXO OXXX XXXX
cocoon cannon saucer	456 309 582 740 605 482 765	OXXOO OXOOO OXOXO
Letter height—1.3748 mm.	*9 Point*	*Acuity: Approx. 0.3 (20/65) Magazines.*
arrow scour noose razor	35 98 20 82 47 30 74 73 42 65 27	XXXO OOXX OXOOX XOXOX
zone reverence sorceress	740 203 965 423 807 203 460 244	XOOXX OOXOO OOOOX OXOX
Letter height—1.3970 mm.	*8 Point*	*Acuity: Approx. 0.4 (20/50) Newspaper text*
amaze wares curve scarce	82 34 65 90 58 83 67 46 98 65 42 54	OXXO OOOO XOXX XXXX OOOX
snooze caress sewer wax	426 397 564 752 205 350 575 369 246	XXXOX XXOOX OOXXO XOXOX
Letter height—1.0414 mm.	*6 Point*	*Acuity: Approx. 0.5 (20/40) Telephone directory.*
comma worse reason measure vase	75 23 68 90 44 63 40 33 82 94 79 90 47 35	XOXX OOXO OXXO OXOO XXOX OOOO
census arrears recover crane now	890 375 204 534 626 987 634 932 246 479 268	XOXXO XXOOO OOOOO OOXOO XOOOX
Letter height—0.8363 mm.	*5 Point*	*Acuity: Approx. 0.6 (20/30) Want ads.*
success numerous assurance consume	56 87 92 30 47 62 45 27 98 80 46 23 43 80	XOXX OOOX OOXO OOXXX XOXO XXOX OXOO
ocean convex morocco uncommon err	209 354 872 405 623 829 204 350 575 482 657	OXOOO OXOXO XXXXO OOXXO XOXXX OXXO
Letter height—0.6985 mm.	*4 Point*	*Acuity: Approx. 0.8 (20/25) Small bibles.*
[illegible]	72 95 40 58 16 55 80 65 30 57 98 46	OXOO XXOX XXXX XXXO XXOO OXOO XOXX OOXO OXX
[illegible]	264 454 308 905 283 460 244 829 350	XOOOX OXXOO OOOXX XXOXX OXXXO OOXXO XXXX
Letter height—0.5207 mm.	*3 Point*	*Acuity: Approx. 1.0 (20/20) Mailorder catalogues.*
[illegible]	[illegible]	[illegible]
Letter height—0.3429 mm.	*2 Point*	*Acuity: Approx. 1.5 (20/13) Photo-reduction.*
[illegible]	[illegible]	[illegible]

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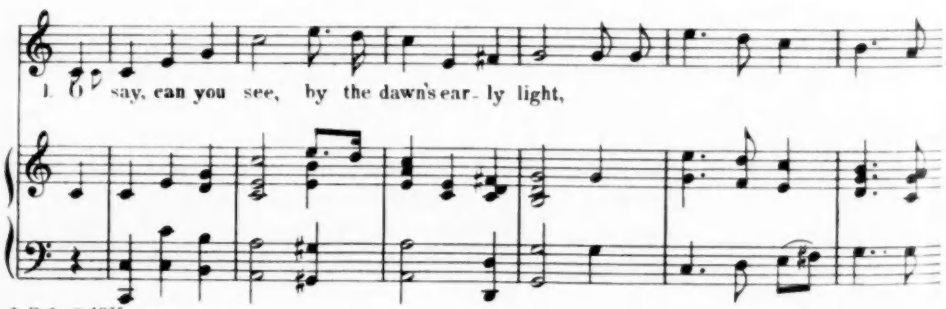
0.6	59	23	E D F C Z P 3746285	20/30	91.4%
0.8	44	17	F E L O P E D 9378668	20/25	95.6%
1.0	35	14	D E F P O T E C 8668795	20/20	100.0%
1.2	29	12	L E F O D P C T 3867954	20/16	
1.5	24	9	C F T L D Z O P 4837	20/13	



J. E. L. © 1935

Letter height—4.2164 mm.	*24 Point*	*Acuity: Approx. 0.12 (20/170) Sight saving texts.*
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Letter height—2.5146 mm.	*14 Point*	*Acuity: Approx. 0.2 (20/100) Books, children 8–9 yrs.*
noon even mew	38 72 80 93 54 60 76	XOXX XXXX OOOX
Letter height—2.0828 mm.	*12 Point*	*Acuity: Approx. 0.25 (20/80) Books, children 9–12 yrs.*
war use worm eve	40 53 82 64 79 60 47	XOOX OOOXO OOXXX
avenue ransom err	809 423 657 980 765	XOXXO XOXOX OXOOX
Letter height—1.7526 mm.	*10 Point*	*Acuity: Approx. 0.28 (20/70) Adult textbooks.*
scum crease nervous	98 67 45 34 23 50 86 73 98	XOOO OXXO OXXX XXXX
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Letter height—1.5248 mm.	*9 Point*	*Acuity: Approx. 0.3 (20/65) Magazines.*
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Letter height—1.0414 mm.	*6 Point*	*Acuity: Approx. 0.5 (20/40) Telephone directory.*
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census arrears recover crane now	890 375 204 534 626 987 654 932 246 479 268	XOXXO XXOOO OOOOO OOXOO XOOOX
Letter height—0.8763 mm.	*5 Point*	*Acuity: Approx. 0.6 (20/30) Want ads.*
success numerous assurance consume	56 87 92 30 47 62 45 27 98 80 46 23 43 80	XOXX OOOX OOXO OOXXX XOXO XXOX OXOO
cornea convex morocco uncommon err	209 354 872 405 625 829 204 350 575 482 057	OXOOO OXOXO XXXXO OOXXO XOXXX OXXO
Letter height—0.6985 mm.	*4 Point*	*Acuity: Approx. 0.8 (20/25) Small bibles.*
[illegible]	72 95 40 58 36 55 80 65 30 57 98 46	OXOO XXOX XXXX XXXO XXOO OXOO XOXX OOXO OXX
	284 458 309 905 283 460 244 829 350	XOOOX OXXOOO OOOOX XXOXX OXXXX OOXXO XXXX
Letter height—0.5207 mm.	*3 Point*	*Acuity: Approx. 1.0 (20/20) Mailorder catalogues.*
[illegible]	[illegible]	[illegible]
Letter height—0.3493 mm.	*2 Point*	*Acuity: Approx. 1.5 (20/13) Photo-reduction.*
[illegible]	[illegible]	[illegible]

Music — Music was included because it is so commonly needed as an empiric test. The 1½ inch staff chosen is frequently found in cheap editions, though the usual height in this country is two inches. The selection is the opening bars of our national air, which so appropriately begins: "O say, can you see, by the dawn's early light,—"

Reading Test — The reading test consists of words composed wholly of short lower case letters such as: wax see now zero arrow cream numerous occurrence nevermore — without any ascending, descending, or dotted characters. There is no context and no variation in contour to suggest clues to the memory. Of the thirteen letters used, Sanford[16] considers w m v the easiest to recognize, r x n u as moderately difficult, and a z o c e s the hardest. Of the eight most frequently occurring letters in English, five are represented by the short letters e a n o s.

The series extends from 24 point Century Schoolbook, the largest size used in books, to 2 point of the same type, which at fourteen inches is beyond the limit of average discrimination. There is thus on the chart an end-point for any individual's reading ability. Each gradation is furnished with data as to its letter height in millimeters; the visual acuity required — noted decimally and in distance equivalent — and its customary use in reading experience. (In rural newspapers 8 point type is customary, but in the city papers 7 point type is generally used. The Chicago Tribune uses 7 point Regal; the Chicago Daily News, 7 point Ideal).

Numbers — The value of numbers in near vision testing has not been sufficiently appreciated. They are international symbols, universally recognized by all who have the least degree of literacy. When standardized, as in this chart, they are the preferred test for the foreigner, especially so if his language is customarily printed in non-roman characters, as with Russian, Greek, Yiddish, and German gothic.

Illiterate Test — The illiterate test consists of four and five letter groups of various combinations of x and o. The illiterate is asked to simply count the "crosses" in each group, as in: oxxo oxoxx. The examination requires only that the child or illiterate be able to count. The reliability of the test has been checked by experiments with literates.

Format — A clean test chart is of the utmost importance, since the loss of contrast in a soiled chart must impair its validity.

The efforts heretofore made to maintain the cleanliness of charts have been only moderately successful. I believe that the format ushered in by this chart solves the problem. A double-faced panel is fashioned by fastening on each side of a six by eight inch piece of plywood a chart, over which a thin sheet of pyralin is welded by heat and pressure. The finished product looks as if covered by a fine clear lacquer. The pyralin panel is durable, shatter-proof, artistically attractive, and easily cleaned; the charts thus processed remain permanently stainless and changeless. In using the charts, a measuring cord attached to an eyelet on the panel's edge is secured by a hook to the endpiece of the patient's glasses or trial-frame, and when taut keeps the card at the required distance. For further convenience, there is affixed to the other edge of the panel a metal pointer.

Procedure — A few hints may be apropos as to the many uses of the chart. The distance vision is noted; *then the brightness of the near vision chart is adjusted to a standard of ten candlepower*, preferably with the aid of a light-meter. In the systematic examination, the following outline is suggested:

I. Using Optotypes

A. Without distance correction, examine for

1. Near Visual Acuity (nV) — This often provides an immediate basis for the judgment of the patient's refraction, since normally, and in most cases of ocular disease, the near visual acuity corresponds to that for remote vision (rV); but it is greater in myopia, less in hypermetropia and presbyopia. Even in presbyopes an idea of the distance refraction is obtained through the difference between their actual near vision and that to be normally anticipated.

2. Far Point of Myopia — With patient fixing on optotype 0.6, carry chart away to the point of blurring. If this distance is beyond 22-23 inches (average norm), continue with the patient fixing on the next larger optotype, and so on.

3. Range of Accommodation — In dealing with mild myopia, hypermetropia, or presbyopia, a plus 3 or 4 diopter sphere is placed before the eye, as suggested by Duane, and the near point and far point measured in diopters on a Prince's rule, measurement to start from the anterior focal point, 14 mm. in front of the cornea. The difference is the range of accommodation. This

method is also recommended for testing the adequacy of cycloplegia.

4. Estimation of visual acuity as a substitute for the distance test:

(a) With hospital, house, or bed cases, where it is impractical to set up or utilize a distance chart, the progressive changes in visual acuity can be regularly recorded with the reduced optotype.

(b) In the office, when the distance vision is below 20/200 — Instead of having the patient walk to the chart, the distance equivalent can be determined at fourteen inches. Vision as low as 20/800 — which is equivalent to counting fingers at two feet — can be thus conveniently measured.

B. With distance correction, can be demonstrated:

1. Near Point of Accommodation — Optotype 1.5 (average norm, 9 inches; 24 cm.) is adapted for this purpose.

2. Presbyopia — Reduction in near visual acuity parallels the recession of the near point. The decline duplicates that section of Donder's curve extending beyond 35 cm. Roughly, a reduction of normal distance acuity to 20/30 or 20/40 equivalent for near means 1.00 D. of presbyopia; and each succeeding notation represents a half diopter more. Thus a reduction to 20/50 equivalent means about 1.50 D. addition; to 20/65, 2.00 D.; to 20/100, 2.50 D.; to 20/200, 3.00 D. With the proper addition, the acuity finally attained should equal that for distance, or be only slightly less.

3. Value of Increased Illumination — That "more light means better sight" is readily proven by the optotype chart and the variable illuminator. With the vision at 1.0 (20/20 equivalent) at 10 candle-power brightness, it reaches to 1.5 (20/13 equivalent) with an increase to 80 candle-power.

4. Malingering — In simulation there is apt to be marked differences between the visual acuity acknowledged for distance, and that to be correspondingly expected for near.

5. Visual Efficiency — The formula for total visual efficiency proposed by the American Medical Association is:

$$\frac{\text{rV.E.} + 2\,\text{nV.E.}}{3}$$

To illustrate: A myope with —9.75 S. as the best correction has a distance acuity (rV) of 20/40; with —6.00 S. the visual acuity for near (nV) is 20/30 equivalent. The total visual efficiency is, hence,

$$\frac{83.6 + 2(91.4)}{3} = 88.8\%$$

In medico-legal work this chart should be a useful supplement to the A.M.A. rating card, as it shows certain gradations not present in the other.

Though the Jaeger series merits but historical interest, many industrial boards still require that near vision be recorded according to the Jaeger classification. What the Jaeger numbers mean in modern printing nomenclature is indicated in the following table:

Jaeger series	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Point system (Century Schoolbook)	3½	4½	6	7	7½	8	10	12	12½	13	14	16	18	24

II. Using Point-Type Chart, provides a basis for

1. Judgment of Patient's Reading Ability — This does not correspond to the smallest type visible (punctum remotum) but to the punctum optimum, which usually represents a fifty per cent larger visual angle (Snell[8]). Thus to read 6 point comfortably demands that the patient discern at least 4 point at the same distance; to read 24 point (sight-saving texts), at least 16 point should be legible; reading 9 or 10 point (upper class work) requires a minimum of 6 point visual acuity. This divergence is explainable by a study of the reading movements, which indicate that a considerable part of the printed line is imaged, not on the fovea, but upon the parafoveal region (Rothlein[18]).

2. Concluding Test in Presbyopia — In correcting presbyopia, the preliminary examination with optotypes is intended to give only the approximate correction. The final adjustment is determined with the point-type chart. The distance at which the patient does his work is carefully noted. For fourteen inches

or beyond, the attention should be directed to the 4 point type; for less than fourteen inches, the 3 point type may be attempted.

The accuracy and latitude of these charts may suggest further uses.

* * *

I am indebted to Professor Sanford R. Gifford for encouragement, suggestion, and criticism; to the Dr. A. C. Snell, Rochester, N. Y.; the American Type Founders Sales Corporation; and the National Society for the Prevention of Blindness for important data.

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PORTABLE DARK ROOM FOR USE IN THE WARD AND HOME

SIDNEY L. OLSHO, M.D.

PHILADELPHIA

THERE IS some difficulty in making an exact ophthalmoscopic examination in a light or lighted hospital ward, or in a home. The media and fine changes in the retina can be seen very much better in a dark room. As it is not always practical to move patients to such a room, the device illustrated is offered.

It consists of a lady's very short-handled, ordinary, opaque, black umbrella. An excellent quality, black sateen curtain, one yard wide, is attached by means of snaps to the umbrella cir-

cumference with an overlap of several inches. The opening can be thrown open, more or less, as desired. This curtain is detachable but is best left in place. When the umbrella is opened we have a dark tent which is placed over the patient. The umbrella handle may be held by the patient or by a nurse. The examiner must have his head inside the curtain.

This portable dark room makes it possible to make a retinoscopic examination in the ward. It affords a dark chamber also for a patient who is recumbent. It provides a dark room suitable for making an eye examination on patients in the outdoors; for instance, as at a tuberculosis sanitarium. The Portable Dark Room may also be useful to a rhinologist who may desire to transilluminate the sinuses in the ward.

For those not desiring to carry the umbrella, it is suggested that the side curtains be attached to a black umbrella top. The tent can then be placed over any available small-sized umbrella.

THE PRECISION ANGLOMETER

GEORGE E. PARK, M.D.*

CHICAGO

THIS INSTRUMENT,† in use at the present time at Northwestern University, tentatively called the Precision Anglometer, consists essentially of a telescope swinging about a graduated arc with vertical and horizontal adjustments of the telescope to bring its axis to coincide with the visual axis and adjustment toward or from the eye to position the arc center at the visual axis center of the eye which is a point fixed within the socket but not fixed with relation to the eye through which the visual axis always passes during any eye excursion.

The relative length, strength, and leverage of the internal and external recti and the angle of deviation are readily found relative to the visual axes even when the cornea is lying beneath a canthus.

Means for finding the separation of the visual axes, instead of the usual pupillary separation, insures positioning of the lenses before the eyes in accordance with the prescription and no error of centration or prescribed prism will result by reason of a difference between the pupillary and visual axis separation. This feature becomes more important as the error of refraction increases.

The angle between the corneal and visual axes is readily found.

The goal in operating for concomitant squint, or in orthoptic training should be to establish muscular balance of the extraocular muscles so that when the visual axes are fixing at infinity there will be no excessive pull on any muscle or muscles in order to maintain fusion.

To date there have been only a limited number of instruments worthy of consideration which are sufficiently accurate for mak-

* From the Department of Ophthalmology, Northwestern University, Chicago, Ill.

† This instrument was designed and constructed by Mr. Clile C. Allen to whom we are greatly indebted.

ing such measurements of the different angles of reflections and deviations to form any basic facts to start with in establishing a law which is to be followed. The Stevens tropometer is about the only instrument which has been used in attempting to determine the relative strength of the individual recti muscles. In it was used a tangent scale which could be set at variable distances from the eye being measured which would necessarily give faulty readings by reason of the varying distances of the scale from the eye.

FIG. 1 — (Park).

It is believed that the data which we are able to obtain with this instrument will eventually make possible a gauged and relatively exact operation in cases of concomitant squint. Not only will we be able to determine the relative length, strength, and leverage of the external and internal recti muscles in order to know the exact amount of advancement or recession which

will be required in order to give the eyes perfect balance, but a large amount of other important information is made available by the instrument which is to be mentioned while describing the technique of its use.

Design — This instrument, which is in use at the present time in the Department of Ophthalmology at Northwestern University, was designed and constructed by Mr. Clile C. Allen[1] to embody the results of research conducted by the author and Russell Smith Park, Professor of Mathematics, Eastern Kentucky Teachers College.[2]

Theory — This research disclosed a fixed point within the orbit which is fixed with relation to the head, but not fixed with relation to the eye, through which the visual axis passes during all horizontal excursions of the eye. We have termed this point, which lies within the eye, the Visual Center of the eye. We also found that the kinemetic center of ocular rotation is not fixed, but moves along a curve a variable distance, from 1.066 mm. to 0.893 mm. nasalward from the visual axis and always on a line perpendicular to the visual axis at the Visual Center. Also there is a variable distance from 14.732 mm. to 13.918 mm. to 12.950 mm., from the point where the visual axis passes through the anterior surface of the cornea to the Visual Center as the angle of the visual axis takes the positions of 39° nasalward, 4° nasalward, and 38° templeward, respectively.

As the visual axis swings upon this point, as a center, during an excursion of the eye, it logically follows that the angular amount of such excursion should be measured with reference to this center.

The importance of the visual center is evident in another respect, viz., that the visual axis seldom intersects the center of the pupil. This being the case, it is evident that any difference between the intercentric distance of the two visual centers and that of the interpupillary distance will result in prism, where no prism is prescribed, due to decentration or, where prism is prescribed, the resultant prism will be of greater or less power than

1. For forty years, Mr. Allen has been a student of optics and mechanics and as a result of his unusual ingenuity he has been able to incorporate his knowledge into this instrument. He has done this work purely for the advancement of science and without any thought of financial gain. It is with sincere appreciation that we acknowledge his keen interest in making this possible.

2. American Journal of Physiology, 104:545-52, (June) 1933.

prescribed, the amount and direction of base of such prismatic error, in either case, being dependent on the strength and sign of the prescribed lenses.

Greater accuracy will be attained by prescribing the intercentric distance of the visual centers for the centering of lenses instead of the usual pupillary distance.

FIG. 2 — (Park).

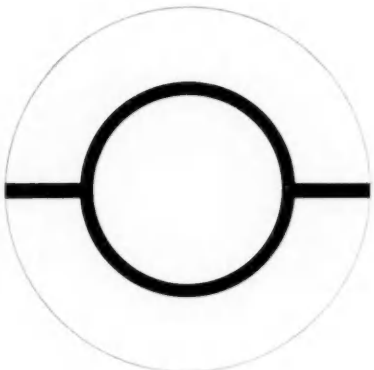


FIG. 3 — (Park).

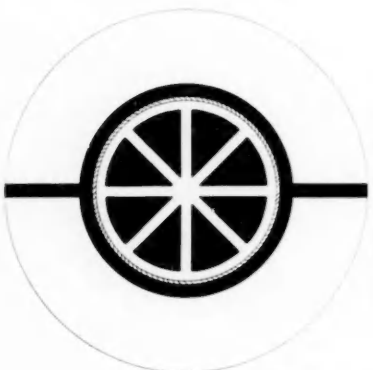


FIG. 4 — (Park).

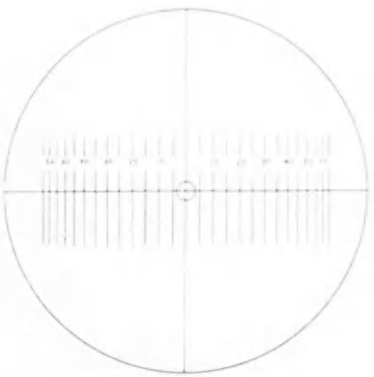


FIG. 5 — (Park).

The Instrument — The instrument, shown in figure 1, is so constructed as to embody the features described. It consists of head support arranged to hold the head firmly in a fixed position facing which is a telescope so arranged that its axis may be brought to coincide with the visual axis of the eye. For this purpose the telescope carries an illuminated target, figure 2, viewed by the patient in establishing this coincidence of axes.

The telescope is movable along a graduated arc in such manner that the vertical projection of the arc center always intersects the telescope axis. Means are provided for bringing this point of intersection to coincide with the visual center of the eye so that all angular measurements of the telescope movement along the arc will be with reference to the visual center.

Means are also provided for determining the separation of the visual centers.

Fixation — The patient is asked to bite a removable wood bit, 1, which is held in slots in the head support by the screw, 2, while elastic pads are brought into contact with the forehead, temples, and back of the head, by their respective screws 3, 4, 5, and 6. By this means the head is firmly held in a fixed position.

The Primary Position — Then bring the telescope to zero on the arc by the knurl 7. Next bring the telescope before the eye horizontally by the knurl 8, and vertically by the knurl 9, until the patient sees a bright spoked wheel, the target, at the center of a dark circle, figure 3, as shown in figure 4. The rim of the wheel is slightly smaller than the dark circle and the patient easily sees when the wheel fits centrally into the dark circle. When this is done, the visual axis coincides with the telescope axis.

Next move the telescope toward or from the eye by the knurl 10, until the image of the target, reflected from the cornea, is seen focused on the eyepiece scale, figure 5, when viewed through the eyepiece. When this is done the visual center of the eye coincides with the intersection of the vertically projected arc center with the telescope axis and all angular measurements of eye movement will be made with reference to the visual center.

The image of the target will not usually lie at the intersection of the central horizontal and vertical lines of the eyepiece scale as the axis of the corneal surface usually lies at an angle to the visual axis. This angle causes an oblique reflection of the target from the corneal surface and its image seen at the eyepiece scale usually lies below and at one side of the center of the scale and is astigmatically distorted. There is always one meridian of the wheel, however, which can be focused in conjunction with the eyepiece scale. This angle between the corneal axis, which is the optic axis of the eye, and the visual axis is the physiological angle. (Physiological angle is used here in-

stead of the much confused nomenclature designated by Angle Alpha, Angle Gamma, or Angle Kappa of Landoldt, since there is but one angle to be considered). This will be discussed in detail in a future publication.

The Physiological Angle — Set the instrument in the primary position, switch on the light in the tube 11, and bring its transparent mirror before the eye vertically by the knurl, 12, and horizontally by the knurl 13, until the patient sees a small red spot superimposed over the center of the target. Then while the patient is directed to fix on this red spot, move the telescope along the arc by the knurl 7, until the center of the target is brought upon the central vertical line of the eyepiece scale. The angle indicated on the arc is the physiological angle as the optic axis of the eye coincides with the telescope axis while the visual axis is held at zero by fixation on the red spot.

Separation of the Visual Centers — Exact determination of this separation is optically necessary only where there is vision in both eyes. Set the instrument in the primary position before one eye and move the scale 15 to indicate zero for this eye. Move the telescope before the other eye by the knurl 8, and secure the primary position for that eye. When this is done the scale indicates the visual center separation.

Where vision is present in but one eye, the scale 15 used in conjunction with the method to be described for securing alignment of the blind eye in 2 and 3, strabismus, or any other method, will suffice as the lens separation is a matter of cosmetics and a separation of the visual centers is unimportant.

Strabismus

1. (Where vision is present in both eyes).

Set the instrument in the primary position before one eye and adjust the tube 11 to where the red spot is seen at the center of the target. Next bring the telescope before the other eye by the knurl 8 and adjust the knurls 8 and 9 for the primary position before this eye.

Again direct the patient to fix the red spot and move the telescope along the arc by the knurl 7 until the red spot is again seen at the center of the target. The first eye is now fixing the red spot and the second eye the target. As these two objects are quite dissimilar there is no tendency to fuse and the angle indicated on the arc is the visual angle of deviation.

If it be found unnecessary to move the telescope from zero on the arc to center the red spot on the target, we know we have orthophoria at infinity. But if there be a tropia or phoria present, the red spot will not be superimposed on the target and must be brought to superimposition on the target by the knurl 7 to determine the angular amount of the tropia or phoria as indicated on the arc. Means are not provided for measuring hyperphorias or hypertropias.

2. (Where vision is present in but one eye).

Set the instrument in the primary position before the good eye and adjust the tube 11 to where the red spot is seen at the center of the wheel. Bring the telescope before the other eye and direct the patient to look to one side in the direction to bring the observed eye approximately into alignment with the telescope. By slight variations in the directions of the patient's gaze and adjustment of the knurls 7 and 8, bring the reflected corneal image of the target to the same distance from the central vertical line of the eyepiece scale as it appeared for the first eye but on the opposite side of the line. Direct the patient to look again at the red spot and move the telescope along the arc by the knurl 7 until the reflected corneal image is again seen in the position just described. The angle indicated on the arc is now the angle of deviation.

3. (Where the pupil of the deviating eye lies under a canthus).

Proceed next as described above for the good eye and for alignment of the second eye with the telescope. In cases where this alignment cannot be effected, the practitioner must use his judgment in locating the telescope before the deviating eye. The telescope being so positioned, note the position of the corneal margin opposite the canthus on the eyepiece scale and direct the patient to look again at the red spot. Note the position to which the corneal margin has moved across the scale. The angle of deviation is the sum of the angular amounts noted on each side of the center of the scale.

The graduations of the scale must not be confused with those of the arc, as those of the scale are used only in the condition just described, in all other observations the arc is used.

Strength of the Recti Muscles—Set the instrument in the primary position, switch off the light in the tube 11, and move it

to one side and down out of the way. Illuminate the eye orbit by moving the bar 14 to the right. Direct the patient to fix the center of the target and move the telescope along the arc by the knurl 7 until an angle is reached where the patient can no longer steadily fix the target. The correct angle is easily found by observing through the telescope just where the eye begins to waver and can fix but momentarily. This gives rotating power of the individual muscle in degrees of arc, from which information is obtained as to the relative length, strength, and leverage of this muscle. If the pupil of the deviating eye lies under a canthus following the rotation we then make the measurements as described under Strabismus 3.

Conclusions

A. An instrument is described which, it is believed, allows for greater accuracy in measuring the ocular movements than is possible with previous instruments.

B. This accuracy depends on the fact that the telescope axis coincides with the visual axis and the center of its arc of rotation corresponds with what we have termed the Visual Center of the eye. This is brought about by the construction of the instrument and by an accurate device to insure fixation at all times. As a matter of interest, the telescope of Gullstrand[3] also incorporates a target which is reflected from the cornea for the sole purpose of observing irregularities of the corneal surface but is not provided with means for measuring angular movements of the eye.

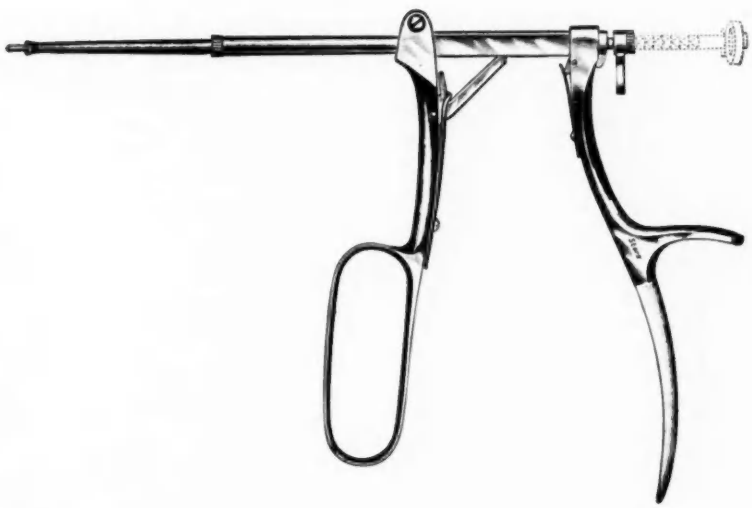
C. Aside from its advantages for scientific investigation of the ocular movements, it is adapted for clinical use. After becoming familiar with its use, a complete record of the horizontal movements may be made in five to ten minutes. Measurements of the vertical movements would require a more complicated instrument than is considered practical at this time.

3. Gullstrand, Allvar: *Photographisch-ophthalmometrische und Klinische Untersuchungen über die Hornhautrefraction*, 1896.

SAGE TONSIL SNARE*

RUSSELL A. SAGE, M.D.

INDIANAPOLIS



A SIMPLE ratchet snare, similar to the Brown and Tyding, of a strong, all-steel construction.

The stilet is round bar with saw-teeth on one side only. A quarter turn releases the stilet easily and quickly from the ratchet to be either pushed forward or withdrawn completely.

A much longer wire than usual can be used without any danger of it being too long to complete the enucleation. The loop can be drawn up gradually and without any possible forward slipping of the wire. The pumping motion used with these handles greatly diminishes the effort on the part of the operator.

* Made by Storz Instrument Company, St. Louis, Mo.

A NEW NEEDLE HOLDER*

D. L. Tilderquist, M.D.

Duluth, Minn.

The main idea in designing this new needle holder was to provide a handle of such size and shape that the instrument could be grasped securely with ease and comfort, thereby forestalling any unexpected slipping or rotation. The other parts are small enough for the most delicate manipulations. The needle end is tapered so that the suture will slip over it smoothly if the instrument is used for tying the knot.

*Made by V. Mueller & Co., Chicago, Ill.

Minutes

The American Academy
of Ophthalmology and Otolaryngology

* * *

Fortieth Annual Meeting
Cincinnati • 1935

MINUTES

AMERICAN ACADEMY *of* OPHTHALMOLOGY *and* OTOLARYNGOLOGY

* * *

MONDAY, SEPTEMBER 16, 1935, 9:30 A.M.

Joint Session

THE JOINT SESSION convened at nine-thirty o'clock, Monday morning, September 16, 1935, in the Hall of Mirrors, Netherland Plaza Hotel, Cincinnati, Ohio, the President, Dr. Wells P. Eagleton of Newark, New Jersey, presiding.

PRESIDENT EAGLETON: In opening the fortieth meeting of the American Academy of Ophthalmology and Otolaryngology, the officers and secretaries extend to you a cordial greeting. We hope that each one of you will take part in the scientific sessions and in the formation of policies of the future.

President Wells P. Eagleton read his Presidential Address, Dr. W. Thornwall Davis, Washington, D. C., First Vice-President, presiding.

Dr. Hans Brunner of Vienna, Austria, the Guest of Honor, read his address on "Brain Tumors and the Ear."

The following symposium on "Headache" was presented:

"Headache from the Standpoint of the Otolaryngologist," by Dr. H. H. Vail, Cincinnati, Ohio.

"Headache from the Standpoint of the Ophthalmologist," by Dr. William H. Crisp of Denver, Colorado.

"Headache from the Standpoint of the Neurologist," by Dr. A. R. Vonderahe, Cincinnati, Ohio (by invitation).

The Joint Session adjourned at twelve twenty-five o'clock.

MONDAY, SEPTEMBER 16, 1935, 2:15 P.M.

Section on Ophthalmology

The first meeting of the Section on Ophthalmology convened at two-fifteen o'clock, Monday afternoon, September 16, Dr. W. Thornwall Davis presiding.

Dr. F. Bruce Fralick, Ann Arbor, Michigan, read a paper (written in conjunction with Dr. Max M. Peet) entitled "The Hypertensive Fundus Oculi After Resection of the Splanchnic Sympathetics." Discussed by Drs. A. D. Ruedemann, Cleveland, Ohio; F. B. Blackmar, Columbus, Georgia; E. M. Josephson, New York, N. Y., and Henry P. Wagener, Rochester, Minnesota.

Dr. Raymond L. Pfeiffer, New York, N. Y., (by invitation), read a paper entitled "Roentgenographic Diagnosis of Retinoblastoma." Discussed by Drs. A. B. Reese, New York, N. Y., and Mary K. Asbury, Cincinnati, Ohio.

Dr. A. B. Reese, New York, N. Y., read a paper entitled "Massive Retinal Fibrosis in Children." Discussed by Drs. Mary K. Asbury, Cincinnati, Ohio, and E. M. Josephson, New York, N. Y.

Dr. Hans Barkan, San Francisco, California, read a paper entitled "Ophthalmic Errors." Discussed by Drs. M. Hayward Post, St. Louis, Missouri; Oscar Wilkinson, Washington, D. C., and E. M. Josephson, New York, N. Y.

Dr. H. W. Woodruff, Joliet, Illinois, read a paper entitled "Management of Complications in Surgery of Senile Cataract." Discussed by Drs. John O. McReynolds, Dallas, Texas; Frank E. Burch, St. Paul, Minnesota; Hans Barkan, San Francisco, California; Oscar B. Nugent, Chicago, Illinois; Georgiana Theobald, Oak Park, Illinois; Richard J. Tivnen, Chicago, Illinois; S. B. Muncaster, Washington, D. C., and J. G. Huizinga, Holland, Michigan.

The Monday afternoon session adjourned at five-fifty o'clock.

MONDAY, SEPTEMBER 16, 1935, 8:30 P.M.

Teachers' Section

The Teachers' Section convened in the Pavilion Caprice, Netherland Plaza Hotel, Cincinnati, Ohio, at eight-thirty o'clock, the chairman of the section, Dr. A. D. Ruedemann of Cleveland, Ohio, presiding.

The discussion was opened by Dr. Wells P. Eagleton, President of the Academy.

A report on the Results of Questionnaire on Graduate and Postgraduate Education in Ophthalmology and Otolaryngology was presented by Dr. Ralph A. Fenton of Portland, Oregon, Secretary of the Section.

The next subject for discussion was "Deficiencies in Training in Anatomy and Pathology in Ophthalmology and Otolaryngology," which was discussed by Dr. Bernard Samuels, New York, N. Y.; Mr. Edward Burchell, New York, N. Y., and Dr. Joseph C. Beck, Chicago, Illinois.

The proposed central laboratory for otolaryngology was discussed by Dr. Frank Novak, Chicago, Illinois.

The subject of "Existing and Proposed Libraries for Ophthalmology and Otolaryngology" was discussed by Dr. Burt R. Shurly, Detroit, Michigan.

Major Raymond O. Dart, Curator of the Army Medical Museum, spoke on "The Army Medical Museum and the Academy."

Dr. Hans Brunner of Vienna, Austria, the Guest of Honor, discussed teaching problems in Vienna.

The report of the Nominating Committee was presented by Dr. S. J. Kopetzky of New York, N. Y., Chairman of the Committee, and the following officers were elected:

Chairman

Dr. Lee W. Dean, St. Louis, Missouri

Vice Chairman

Dr. J. H. Dunnington, New York, N. Y.

Members of Board of Directors
to serve for ensuing five years
Dr. A. D. Ruedemann, Cleveland, Ohio
Dr. George M. Coates, Philadelphia, Pennsylvania

Member of Board of Directors to fill vacancy
left by resignation of Dr. Thomas Allen
Dr. C. S. O'Brien, Iowa City, Iowa

The meeting adjourned at ten-fifteen o'clock.

TUESDAY, SEPTEMBER 17, 1935, 2:00 P.M.

Section on Otolaryngology

The first meeting of the Section on Otolaryngology convened at two o'clock, Tuesday afternoon, September 17, the Second Vice President, Dr. Samuel Iglauer of Cincinnati, Ohio, presiding.

Dr. Ralph A. Fenton, Portland, Oregon, read the Research Report on Experimental Sinusitis (Research Grant by the Council), which was written in conjunction with Dr. Olof Larsell.

Dr. Warren T. Vaughan, Richmond, Virginia (by invitation), read a paper entitled "Allergy in the Rhinologist's Waiting Room." Discussed by Drs. French K. Hansel, St. Louis, Missouri; Hugh S. Kuhn, Hammond, Indiana, and Herman Semenov, Los Angeles, California.

Dr. Lyman H. Heine, Boston, Massachusetts, read a paper entitled "The Effect of Radiation upon Ciliated Epithelium." Discussed by Drs. Harris P. Mosher, Boston, Massachusetts, and Ralph A. Fenton, Portland, Oregon.

Dr. Samuel Salinger, Chicago, Illinois, read a paper entitled "Malignancies of the Epipharynx" (prepared in conjunction with Dr. S. J. Pearlman). Discussed by Dr. Gordon B. New, Rochester, Minnesota.

Dr. Oscar V. Batson, Philadelphia, Pennsylvania, read a paper entitled "Use of Gelatin Prostheses in Facial Restoration." Discussed by Drs. Samuel Iglauer, Cincinnati, Ohio; Ferris Smith, Grand Rapids, Michigan; Francis Lederer, Chicago, Illinois, and Joseph Beck, Chicago, Illinois.

The Tuesday afternoon session adjourned at five o'clock.

WEDNESDAY, SEPTEMBER 18, 1935, 2:00 P.M.

Section on Ophthalmology

The second meeting of the Section on Ophthalmology convened at two o'clock, Wednesday afternoon, September 18, Dr. W. Thornwall Davis presiding.

Dr. Wendell L. Hughes, Hempstead, Long Island, N. Y., read a paper entitled "Aniseikonia: Some Clinical Applications." Discussed by Dr. Walter B. Lancaster, Boston, Massachusetts.

Dr. James W. White, New York, N. Y., read a paper entitled "Congenital Paralysis of the Sixth Nerve." Discussed by Drs. John F. Gipner, Rochester, New York; Meyer Wiener, St. Louis, Missouri, and L. C. Peter, Philadelphia, Pennsylvania.

Dr. Don Marshall, Ann Arbor, Michigan, read a paper entitled "Changes in Refraction Following Operation for Strabismus." Discussed by Drs. J. L. Bressler, Chicago, Illinois, and Oscar Wilkinson, Washington, D. C.

Dr. L. A. Julianelle, St. Louis, Missouri, read a paper (prepared in conjunction with Dr. R. W. Harrison) entitled "Studies on the Infectivity of Trachoma." Discussed by Drs. Phillips Thygeson, Iowa City, Iowa, and M. M. Cullom, Nashville, Tennessee.

Dr. Harry S. Gradle, Chicago, Illinois, read a paper (prepared in conjunction with Dr. A. L. Lenzen and Miss Aubrey Hayden) entitled "The Trachoma Situation in Southern Illinois." Discussed by Dr. Richard C. Gamble, Chicago, Illinois.

The Wednesday afternoon session adjourned at five o'clock.

WEDNESDAY, SEPTEMBER 18, 1935, 7:00 P.M.

Banquet

The Banquet proved to be a very enjoyable party. It was held in the beautiful Hall of Mirrors, Netherland Plaza Hotel.

The occasion was distinctive in that it was the seventieth birthday of President Eagleton, who presided.

The following were honored guests: Dr. Hans Brunner, Vienna, Austria, Academy Guest of Honor; Dr. Hal Foster, Kansas City, Missouri, the first Secretary of the Western Ophthalmological Society; Mrs. Derrick Vail, and Dr. Alfred Friedlander, Dean, Medical School, University of Cincinnati, Cincinnati, Ohio.

Dr. M. A. Goldstein, St. Louis, Missouri, the oldest living past President of the Academy, gave his address, "The History of the American Academy of Ophthalmology and Otolaryngology."

Installation ceremonies for the President-Elect, Dr. Frank E. Burch, St. Paul, Minnesota, were held.

This was followed by dancing.

THURSDAY, SEPTEMBER 19, 1935, 2:00 P.M.

Section on Otolaryngology

The second session of the Section on Otolaryngology convened at two o'clock, the President, Dr. Wells P. Eagleton, presiding.

The Report on Tinnitus (research grant by Council) was read by Dr. William J. McNally, Montreal, Canada, (written in conjunction with Drs. E. A. Stuart, T. F. Reid, and L. H. McConnell). Discussed by Dr. L. K. Guggenheim, St. Louis, Missouri.

Dr. Dean M. Lierle, Iowa City, Iowa, read a paper entitled "Focal Infection in Arthritis — A Statistical Study." Discussed by Drs. John J. Shea, Memphis, Tennessee, and Henry M. Goodyear, Cincinnati, Ohio.

Dr. Cecil Striker, Cincinnati, Ohio, (by invitation) read a paper entitled "The Relation of Diet to Certain Otolaryngologic Problems." Discussed by Drs. Francis Blackmar, Columbus, Georgia, and Ira Frank, Chicago, Illinois.

Dr. M. H. Lurie, Boston, Massachusetts, read a paper entitled "Animal Experimentation on Hearing — Its Clues to the Prevention of Deafness." Discussed by Drs. H. P. Mosher, Boston, Massachusetts, and L. K. Guggenheim, St. Louis, Missouri.

Dr. Gabriel Tucker, Philadelphia, Pennsylvania, read a paper entitled "Observations on Chronic Inflammatory Lesions of the True Vocal Cords." Discussed by Drs. Ellen J. Patterson, Pittsburgh, Pennsylvania, and M. F. Arbuckle, St. Louis, Missouri.

The final scientific session adjourned at five o'clock.

THURSDAY, SEPTEMBER 19, 1935, 8:10 P.M.

"Medical Economics"

A meeting for the discussion of "Medical Economics" was held at eight-ten o'clock, the President, Dr. Wells P. Eagleton, presiding.

An address on "Medical Economics" was given by Dr. R. G. Leland, Director of the Medical Economics Bureau of the American Medical Association, Chicago, Illinois.

This address was discussed by the following members of the Academy: Drs. Gordon Harkness, Davenport, Iowa; Grover C. Otrich, Belleville, Illinois; Howard V. Dutrow, Dayton, Ohio; H. H. Vail, Cincinnati, Ohio; H. S. Gradle, Chicago, Illinois; Burt R. Shurly, Detroit, Michigan; Lee W. Dean, St. Louis, Missouri, and Wells P. Eagleton, Newark, New Jersey. Dr. Leland closed the discussion.

The meeting adjourned at ten-thirty o'clock.

THURSDAY, SEPTEMBER 19, 1935, 5:30 P.M.

Business Meeting

The annual Business Meeting of the American Academy of Ophthalmology and Otolaryngology convened at five-thirty o'clock, Thursday afternoon, the President, Dr. Wells P. Eagleton, presiding.

The following reports were read:

REPORT OF EXECUTIVE SECRETARY-TREASURER

The membership summary as of September 1, 1935, is as follows:

Honorary Members	19	
Life Members	116	
Senior Members	98	
Junior Members	2,013	
		2,246
Candidates for membership		163
Total carried on books		2,409

During the year 1935, thirty-five deaths were reported, which will be summarized in the report of the Historian.

Members delinquent in dues total as follows:

1935—Senior Members	3
Junior Members	103
1934 and 1935	39
1933, 1934, and 1935	67
1932, 1933, 1934, and 1935	52

The Council at this session conferred Honorary memberships on:

Dr. Hans Brunner, Vienna, Austria
Dr. Hal Foster, Kansas City, Missouri

Elevated to Life Membership:

Dr. A. L. Adams, Jacksonville, Illinois
Dr. C. F. Adams, Trenton, New Jersey
Dr. Robert Beattie, Detroit Michigan
Dr. C. H. Brobst, Peoria, Illinois
Dr. T. A. Burke, Cleveland, Ohio
Dr. W. W. Carter, New York, N. Y.
Dr. J. Leslie Davis, Philadelphia, Pennsylvania
Dr. Linn Emerson, Orange, New Jersey
Dr. E. S. Ferguson, Oklahoma City, Okla.
Dr. I. Grushlaw, New York, N. Y.

Dr. C. C. Hetzel, Ogden, Utah
Dr. H. B. Hitz, Milwaukee, Wisconsin
Dr. G. L. King, Alliance, Ohio
Dr. John F. Klinedinst, York, Pennsylvania
Dr. R. S. Lamb, Washington, D. C.
Dr. Charles Lukens, Toledo, Ohio
Dr. R. E. Mercer, Detroit, Michigan
Dr. J. W. Millette, Dayton, Ohio
Dr. C. L. Minor, Springfield, Ohio
Dr. John H. Rindlaub, Fargo, North Dakota
Dr. H. H. Sanderson, Detroit, Michigan
Dr. R. D. Sleight, Battle Creek, Michigan
Dr. W. H. Snyder, Toledo, Ohio
Dr. E. S. Strout, Minneapolis, Minnesota
Dr. C. A. Veasey, Spokane, Washington

Elevated to Senior Membership:

Dr. D. F. Berry, Indianapolis, Indiana
Dr. J. M. Brown, Los Angeles, California
Dr. J. M. Garber, Mansfield, Ohio
Dr. H. M. Goddard, Philadelphia, Pennsylvania
Dr. L. J. Goldback, Baltimore, Maryland
Dr. H. S. Gradle, Chicago, Illinois
Dr. Allen Greenwood, Boston, Massachusetts
Dr. Gordon F. Harkness, Davenport, Iowa
Dr. M. David Haspel, New Orleans, Louisiana
Dr. Wilfred H. Haughey, Battle Creek, Michigan
Dr. Carl H. McCaskey, Indianapolis, Indiana
Dr. J. L. McCool, San Francisco, California
Dr. William F. Molt, Indianapolis, Indiana
Dr. F. V. Overman, Indianapolis, Indiana
Dr. H. L. Pollock, Chicago, Illinois
Dr. M. Ravdin, Evansville, Indiana
Dr. George S. Row, Indianapolis, Indiana
Dr. W. Likely Simpson, Memphis, Tennessee
Dr. Henry Stanbery, Cincinnati, Ohio

The following resignations have been accepted:

Dr. J. B. Wright, Raleigh, North Carolina
Dr. Samuel Wolf, Chicago, Illinois
Dr. Gustave Bruere, Portland, Oregon

In spite of the depression, no change has been made in the policy of expansion and operation. Obviously, the delinquent percentage in dues has lowered the operating income; however, for the year 1934, the operating surplus amounted to $2,384.92. The associate secretaries serving without remuneration have made this surplus possible.

The Academy has rapidly increased its influence in the field of medicine. The Council and the Board of Secretaries considered as the management appreciate the responsibility imposed.

The report of the auditor for the year 1934 is appended as a part of this report. Summarizing, briefly, during 1934 the income per member from dues and other sources was $16.73; the cost was $15.00 per member. Of the disbursements, $3,500.00 was accounted for by research grants.

For the current year 1935, from an audit of the first eight months (to September 1, 1935), and estimating the income and disbursements for the balance of the year, the Academy has improved its financial status over that of the corresponding period (up to the Chicago convention of 1934).

The deaths of Dr. Mullin and Dr. Carmack, the latter, although without title, was a part of the secretarial group, forced the realization that others should be trained to step into official activities in case of sudden changes. The Council has been asked to provide for such emergencies.

On behalf of the Secretarial Group, of which I am only a part, I do desire to express appreciation for the splendid cooperation of the President and the other officers during the current year.

Respectfully submitted,

W. P. Wherry, M.D.

Report of the Auditor

Omaha, Nebr., January 2, 1935

The American Academy of
Ophthalmology and Otolaryngology

Gentlemen:

I have examined the accounts of your Academy for the period from January 1, 1934, to December 31, 1934. I submit herewith a summary of receipts and disbursements, together with a statement of assets at December 31, 1934.

All receipts and disbursements were traced to the Academy's bank account, of necessity, some cash received at the Chicago Convention was disbursed.

The ordinary general expenses for the month of December have not been paid.

Office furniture and instruments are not listed among the assets.

The bonds owned by the Academy are in possession of the Trust Department of the Omaha National Bank for safekeeping. They were verified by a certificate from the Bank. These securities are carried at cost, with no adjustments for amortization of premium or discount. Interest coupons maturing amounted to $5,440.00, of which $3,923.95 was received, leaving $1,516.05 in default.

The changes in the financial status of the Academy during the year are accounted for as follows:

Assets held January 1, 1934		$107,518.40
Add: Excess of Receipts over Disbursements:		
Receipts	$34,060.61	
Disbursements	31,051.74	3,008.87
Assets December 31, 1934		$110,527.27

I certify that the Statements are correct to the best of my knowledge and belief.

JOHN P. BEGLEY,
Certified Public Accountant

RECEIPTS AND DISBURSEMENTS

JANUARY 1, 1934, TO DECEMBER 31, 1934

Cash Balance January 1, 1934		$ 1,736.68
Receipts:		
Applications, dues, etc.	$18,900.00	
Transactions	977.01	
Convention	10,259.65	
Bond interest	3,923.95	34,060.61
On Capital Account:		
Bonds sold	None	
		$35,797.29

Disbursements:

Research	$ 3,300.00	
Transactions	8,329.65	
Honorariums	2,250.00	
Meetings and conventions	11,510.46	
Office and general expense	5,661.63	31,051.74
On Capital Account:		
Bonds purchased	None	
		$ 4,745.55

SUMMARY OF ASSETS, DECEMBER 31, 1934

Cash Balance	$ 4,745.55
Securities owned (at cost)	105,781.72
	$110,527.27

In Memoriam

Dr. Kate Wylie Baldwin, Philadelphia, Pennsylvania
Dr. Harold Jolly Beard, Youngstown, Ohio
Dr. John Walter Carmack, Indianapolis, Indiana
Dr. Eugene R. Carpenter, Dallas, Texas
Dr. George Gregory Carroll, Rochester, New York
Dr. George Benjamin Collier, New Orleans, Louisiana
Dr. Arthur Clark Dean, Hot Springs, South Dakota
Dr. Ira Hugh Dillon, Topeka, Kansas
Dr. Oscar Beesley Dunn, Ironton, Ohio
Dr. Herman Frederick William Flock, Williamsport, Pennsylvania
Dr. John William Foley, Denver, Colorado
Dr. John Randolph Garrett, Roanoke, Virginia
Dr. Anthony J. Giordano, New York City, N. Y.
Dr. Edward Baldwin Gleason, Philadelphia, Pennsylvania
Dr. Peter Taylor Grant, Grand Rapids, Michigan
Dr. John Francis Healey, Buffalo, New York
Dr. Frank John Holdsworth, Traverse City, Michigan
Dr. Edward John Hussey, Holyoke, Massachusetts
Dr. George Clyde Kneedler, Pittsburgh, Pennsylvania
Dr. William W. Lewis, St. Paul, Minnesota
Dr. Clarence Loeb, Hubbard Woods, Illinois
Dr. Lewis E. Maire, Grosse Point, Michigan
Dr. Alexander Dunbar McKelvey, Toronto, Canada
Dr. Allyn Bernard Moise, Shreveport, Louisiana
Dr. William Valentine Mullin, Cleveland, Ohio
Dr. Clarence Anson Neighbors, Anaheim, California
Dr. Harry Edward Oesterling, Winter Park, Florida
Dr. William Nelson Offutt, Lexington, Kentucky
Dr. Victor Ray, Sr., Cincinnati, Ohio
Dr. William Scott Renner, Buffalo, New York
Dr. Duncan Campbell Smyth, Boston, Massachusetts
Dr. Morie Frederick Weymann, Los Angeles, California
Dr. Douglas Fox Wood, Minneapolis, Minnesota
Dr. John Westley Wright, Columbus, Ohio
Dr. Henry Byrd Young, Burlington, Iowa
Dr. Edward Frank Ziegelman, San Francisco, California

REPORT OF THE HISTORIAN

In the absence of the Historian, Dr. James A. Babbitt, his report was read by Secretary Wherry, the members standing in silent tribute during the reading of the report.

(The report was accepted and placed on file.)

Dr. Kate Wylie Baldwin

Dr. Kate Wylie Baldwin of Philadelphia, who died on January 18, 1935, was a graduate of the Woman's Medical College of Pennsylvania in 1890.

For many years she was on the staffs of the Woman's and Children's Hospitals, and Demonstrator, Clinical Instructor, and Instructor in Surgery and Adjunct Professor of Surgery at the Woman's Medical College.

Dr. Baldwin was a member of the American Academy of Ophthalmology and Otolaryngology and a Fellow of the American College of Surgeons.

Dr. Baldwin was admired and respected by her contemporaries in otolaryngology. She had such a dignified, keen and alert personality and left a definite impress of latent intellectual strength. Her opinion was valued, and her discussions, though laconic, were a real contribution.

Dr. Harold Jolly Beard

Dr. Harold Jolly Beard was born on January 12, 1880, at Hardinsburg, Kentucky, and died January 30, 1935, at Youngtown, Ohio.

Graduating from Breckinridge Normal College at Hardinsburg, Dr. Beard took his B.S. and medical degrees from the Kentucky School of Medicine at Louisville, Kentucky, and did postgraduate work in New York, Chicago and Boston.

He began the practice of medicine at Evansville, Indiana, in 1901, later moving to Youngstown, Ohio, and taking up the practice of his specialties. At the time of his death, Dr. Beard was on the staffs of the Youngstown City Hospital and St. Elizabeth's Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and past president of the Mahoning County Medical Society.

Dr. Beard is survived by his father, one brother and two sisters.

Dr. John Walter Carmack

Dr. John Walter Carmack was born on March 24, 1885, in Dana, Indiana, and was killed in an airplane accident at Richmond, Indiana, on December 5, 1934.

Dr. Carmack received his medical degree from Indiana University in 1907, beginning the practice of medicine in Indianapolis in 1908, and limiting it to the ear, nose and throat in 1918.

He was Clinical Professor of Otology, Rhinology and Laryngology at the Indiana University School of Medicine, surgeon to the Robert W. Long Hospital, the James Whitcomb Riley Hospital for Children, Indianapolis City Methodist Episcopal, St. Vincent's and Christian Hospitals in Indianapolis.

Dr. Carmack was a member of the American Academy of Ophthalmology and Otolaryngology, the American Laryngological Association, the American Laryngological, Rhinological and Otological Society, a Fellow of the American College of Surgeons, and in 1934 was secretary of the Section on Otolaryngology of the American Medical Association.

He is survived by his wife and son.

Dr. Carmack was one of the real stand-bys of the American Academy of Ophthalmology and Otolaryngology. When in a recent symposium your historian desired the company of two particularly pleasing, earnest and efficient participants in this presentation, he selected and obtained the cooperation of this man. His presence was stimulating, his cheery smiling face was a constant delight. There never was a finer, more clear cut type of otolaryngologist than John Walter Carmack.

Dr. Eugene R. Carpenter

Dr. Eugene R. Carpenter, who died in Dallas, Texas, on October 9, 1934, was born in Knobnoster, Missouri, on October 5, 1873.

After graduating from the University of Michigan in 1894 and Jefferson Medical College, Philadelphia, in 1898, in which latter class he was among the honor graduates and was awarded the de Schweinitz medal in ophthalmology, the Dercum neurological prize and the otological prize, he served an interneship at Kings College Hospital, New York, and later did postgraduate work in the Manhattan Eye and Ear Hospital and the New York Eye and Ear Infirmary, and at medical centers in Vienna and London.

Dr. Carpenter was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Editors' and Authors' Association, Fellow of the American College of Surgeons, past president of the El Paso County Medical Association, honorary member of the New Mexico Medical Association and a member of the State Medical Association of Texas. At the time of his death he was on the staffs of the Baylor, Methodist and Parkland Hospitals.

Dr. Carpenter served fourteen months with the United States Army in the World War and was retired with the rank of captain. At the time of his death he held the rank of major in the Medical Reserve Corps.

He was a designer of several surgical instruments and the author of numerous medical and surgical articles.

His wife, parents, two brothers and a sister survive him.

Dr. George Gregory Carroll

Dr. George Gregory Carroll, a son of Dr. George Goodrich Carroll, was born on May 9, 1874, and died July 23, 1935, in Rochester, New York.

His early education was obtained from the Rochester Free Academy and the University of Rochester, following which he graduated in medicine from the Medical College of the University of Pennsylvania in 1900, and took postgraduate courses in New York City and Vienna. Returning to Rochester, he was inspired by the work of Dr. Chevalier Jackson of Philadelphia to continue his studies in bronchoscopy, and was western New York's pioneer bronchoscopic specialist. In 1924, he established their first bronchoscopic clinic in St. Mary's Hospital.

At the time of his death, Dr. Carroll was president of the staff and member of the board of the Park Avenue Hospital; bronchoscopist there and at St. Mary's, the Genesee and Monroe County Hospitals, and ear, nose and throat surgeon for St. Mary's Boys' Orphan Asylum.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Laryngological, Rhinological and Otological Society, the American Bronchoscopic Society, Fellow of the American College of Surgeons, and diplomate of the American Board of Otolaryngology, also a member of his state and county medical societies.

Dr. Carroll is survived by his mother, a daughter, four sisters, and two brothers.

Dr. George Benjamin Collier

Dr. George Benjamin Collier, of New Orleans, Louisiana, who died on July 9, 1934, at the age of forty-four years, was a graduate of Tulane University of Louisiana School of Medicine, New Orleans, in 1915.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the New Orleans Ophthalmological and Otolaryngological Society, Fellow of the American College of Surgeons, and diplomate of the American Board of Otolaryngology.

At the time of his death Dr. Collier was instructor of anatomy in Senses Department of Tulane University Postgraduate School, Surgeon in Charge of the Ear, Nose and Throat Department of the Eye, Ear, Nose and Throat Hospital, and on the staff of the Southern Baptist Hospital.

Dr. Collier served as Lieutenant in the Medical Corps during the World War.

Dr. Arthur Clark Dean

Dr. Arthur Clark Dean was born July 23, 1887, in Hatton, North Dakota, and died December 1, 1934, in East St. Louis, Illinois.

After receiving his early education at Fargo College Academy and the University of North Dakota, he obtained his medical degree from Jefferson Medical College, Philadelphia, in 1917, following which he immediately entered World War service, first at the Naval Hospital in New York, then the Naval Yard in Philadelphia, and as lieutenant in the Medical Corps on the Hospital Ship Mercy. In July, 1919, he became associated with the Campbell, Wheeler and Williamson Medical Clinic at Grand Forks, and later took postgraduate courses in eye, ear, nose and throat work in Chicago and New York.

From 1926 to 1929, Dr. Dean was head of the Eye, Ear, Nose and Throat Department of the Crookston Clinic, Crookston, Minnesota, later going to the Department of Eye, Ear, Nose and Throat at the Hot Springs Clinic, Hot Springs, South Dakota, and was Consulting Specialist to the Eye, Ear, Nose and Throat Department of the United States Veterans Hospital at Battle Mountain Sanitarium, Hot Springs, South Dakota, also physician and surgeon to the Great Northern, Chicago and Northwestern

Railway. In September, 1934, he took over a private practice at East St. Louis, Illinois, and at the time of his death was on the teaching staff of Washington University and Oscar Johnson Institute, St. Louis, Missouri.

Dr. Dean was a member of the American Academy of Ophthalmology and Otolaryngology, and a Fellow of the American College of Surgeons. He was the author of many papers on medical subjects, and abstract editor for *Minnesota Medicine*.

He is survived by his wife and son.

Dr. Ira Hugh Dillon

Dr. Ira Hugh Dillon was born in Putnam County, Missouri, September 15, 1873, and died in Topeka, Kansas, on November 16, 1934.

Receiving his early education in the schools of Missouri, he entered the medical school at Iowa State University, and received his medical degree from the Illinois State Medical School in 1897. He began to practice medicine in Auburn, Nebraska, in 1898. After postgraduate studies in eye, ear, nose and throat work in 1904 and again in 1915, he limited his practice to those specialties.

Dr. Dillon served for two years in the Army Medical Corps stationed at Fort Worth, Texas, in the Base Hospital at Camp Bowie, where he acted as Chief of Surgery of the Head, first as captain and being promoted to major in 1918.

He served as State Health Commissioner of Nebraska from 1919 to 1923, and then again took up his specialties at Wellington, Kansas. At the time of his death he was on the staff of the Santa Fe Hospital in Topeka, Kansas.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, member of his county and state medical societies, the Sioux Valley Eye and Ear Academy and the Kansas City Eye, Ear, Nose and Throat Society.

He is survived by his wife and two daughters.

Dr. Oscar Beesley Dunn

Dr. Oscar Beesley Dunn died in Ironton, Ohio, on September 11, 1934, aged 84 years.

Graduating from Miami Medical College in Cincinnati in 1878, he practiced his specialty in Ironton for fifty years, retiring from active practice in 1928 following injuries received in an automobile accident.

He was a life member of the American Academy of Ophthalmology and Otolaryngology and a member of the Ohio State Medical Association and the American Medical Association. He also belonged to the Masonic Lodge.

Dr. Dunn is survived by three sisters and one brother.

Dr. Herman Frederick William Flock

Dr. Herman F. W. Flock was born in Williamsport, Pennsylvania, on October 1, 1895, and died on March 7, 1935.

Receiving his early education in the Williamsport schools and his medical degree from Jefferson Medical College, Philadelphia, Dr. Flock took postgraduate work in the Manhattan Eye, Ear and Throat Hospital, and later took special studies in Germany and Austria.

At the time of his death, Dr. Flock was on the staff of the Williamsport Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the Lycoming County Medical Society, Pennsylvania State Medical Society, and a Fellow of the American College of Surgeons.

Dr. Flock is survived by his wife, one daughter and his parents.

Dr. John William Foley

Dr. John W. Foley was born in Rochester, New York, on March 28, 1873, and died in Denver, Colorado, on February 17, 1935.

Receiving his early education in Ann Arbor, Michigan, and his medical degree from the University of Michigan in 1894, Dr. Foley did postgraduate work in Philadelphia, New York and the University of Michigan and research work later at Moorefield Hospital, London, and in Vienna and Berlin.

In 1898 he moved to Denver and took up the practice of his specialties. During the war he served as major, and was stationed at Fort Sam Houston, Texas.

Dr. Foley was a member of the American Academy of Ophthalmology and Otolaryngology, and aside from his medical interests he was keenly interested in music. He was a member of the Knights of Columbus and Elks Clubs.

His widow and two sisters survive him.

Dr. John Randolph Garrett

Dr. John Randolph Garrett of Roanoke, Virginia, who died on August 28, 1934, aged sixty-five, received his medical degree in the University College of Medicine, Richmond, in 1898.

He was a member of the American Academy of Ophthalmology and Otolaryngology and a Fellow of the American College of Surgeons.

At the time of his death, Dr. Garrett was on the staff of the Roanoke Hospital.

He is survived by his widow and one son.

Dr. Anthony J. Giordano

Dr. Anthony J. Giordano was born on March 23, 1897, in New York City, and died in Yonkers, New York, on January 23, 1935.

Dr. Giordano received the degrees of F.A.C.S. and M.D. from Fordham University, New York, in 1920, and began the practice of medicine in New York City in March, 1921. He later began to specialize in ear, nose and throat work and took postgraduate work in Vienna in 1926.

At the time of his death Dr. Giordano was Chief of Staff of the Ear, Nose and Throat Department of St. Joseph's Hospital, Yonkers, and Visiting Surgeon of the Seton Hospital and Columbus Hospital Extension, 167th Street, New York.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Society of Vienna, the Bronx County Medical Society, the Bronx Italian Medical Society, and a Fellow of the American College of Surgeons.

Dr. Giordano is survived by his wife, his mother, two sons, and three daughters.

Dr. Edward Baldwin Gleason

Dr. Edward B. Gleason was born in Philadelphia, on October 13, 1854, and died there on November 30, 1934.

Educated in private schools in Philadelphia, he received his medical degree from the University of Pennsylvania in 1878, and from the Medical and Chirurgical College in 1899. In 1905 Villanova College gave him the honorary degree of LL.B.

Dr. Gleason was formerly professor of otology at the Medico-Chirurgical College of Philadelphia, and laryngologist to the Philadelphia Hospital; surgeon in charge of the nose, throat and ear department of the Northern Dispensary; state's attorney for the Pennsylvania Society, Order of Founders and Patriots, and a surgeon in the Medical Reserve Corps with the rank of first lieutenant.

He was a life member of the American Academy of Ophthalmology and Otolaryngology, the American Otological Society, the Pennsylvania Medical Society, a Fellow of the College of Physicians of Philadelphia and of the American Academy of Medicine.

Dr. Gleason at the time of his death was professor of otology in the Graduate School of the University of Pennsylvania. His instruction to graduate students in dissections about the ear was an exhilarating course. Gleason was a most genial and kindly spirit. Few people had a larger circle of friends both lay and professional. His interests were manifold — at one time even including political activity in his native city. He was author of the "Manual of Diseases of the Nose, Throat and Ear."

He is survived by his wife and one daughter.

Dr. Peter Taylor Grant

Dr. Peter Taylor Grant was born in Parksville, New York, on March 7, 1884, and died in Grand Rapids, Michigan, on July 16, 1934.

He received his medical degree from the Kentucky School of Medicine and began the practice of medicine in Marengo, Indiana; later doing special postgraduate studies in his specialties in New York, Philadelphia, and Vienna, and practicing in Grand Rapids.

Dr. Grant was a member of the American Academy of Ophthalmology and Otolaryngology, local and state medical societies,

and at the time of his death was Chairman of the Section on Otolaryngology of the Michigan State Medical Association and Vice President of the Detroit Otolaryngological Association.

Dr. Grant was a Mason, and a member of the Peninsular and Lions Clubs.

He is survived by his widow and three daughters.

Dr. John Francis Healey

Dr. John Francis Healey was born in Elmira, New York, on April 9, 1893, and died in Buffalo, New York, on November 30, 1934.

He received his medical degree from Buffalo University and did postgraduate work in New York Postgraduate Hospital, following which he took up the practice of ear, nose and throat work in Buffalo in 1918.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association and the Erie County Medical Society.

At the time of his death Dr. Healey was on the staffs of Buffalo City Hospital, Buffalo General Hospital and the Children's Hospital.

He served during the World War.

His daughter, mother and two sisters survive him.

Dr. Frank John Holdsworth

Dr. Frank John Holdsworth of Traverse City, Michigan, who died on April 4, 1935, at the age of sixty years, was graduated from the University of Michigan in 1900, and began the practice of medicine in that year in Williamsburg, Michigan. Later he took up the practice of his specialties in Traverse City.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and the Michigan State Medical Society, and at the time of his death was on the staff of the James Decker Munson Hospital and Consultant at the Michigan State Hospital.

His widow, one son, Dr. M. G. Holdsworth, and two grandchildren survive him.

Dr. Edward John Hussey

Dr. Edward John Hussey was born in Holyoke, Massachusetts, on June 29, 1883, and died there on December 4, 1934.

Graduating from the Harvard Medical School in 1903, Dr. Hussey interned at Carney Hospital and the Eye and Ear Infirmary of the Massachusetts General Hospital, and later studied abroad.

After serving in the World War at the Mesves Hospital on the Loire and later at the Luxemburg City Evacuation Hospital with the rank of captain, he returned to this country and was in the United States Army Reserve Corps with the rank of lieutenant colonel.

Dr. Hussey was a member of the American Academy of Ophthalmology and Otolaryngology, the New England Ophthalmological Society and the New England Otological and Laryngological Society, and a Fellow of the American College of Surgeons.

At the time of his death he was Chief of Staff in the eye and ear division of the Holyoke and Providence Hospitals, and was president of the staff of the latter.

He is survived by his widow, a stepdaughter, and one sister.

Dr. George Clyde Kneedler

Dr. George Clyde Kneedler was born in Cherry Tree, Indiana County, Pennsylvania, on February 22, 1868, and died in Bellevue, Pennsylvania, on October 12, 1934.

He graduated from Valparaiso University with the degree of A.B., and won his medical degree from the University of Pittsburgh, after which he took postgraduate ear, nose and throat work in New York, Chicago, and Berlin, Germany. Beginning medical practice in 1892, he confined his efforts to his specialty of the ear, nose and throat since 1910.

During the early part of his special practice Dr. Kneedler was affiliated with the Eye and Ear Hospital of Pittsburgh and later was on the staff of the Suburban General Hospital, Bellevue, Pennsylvania.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Board of Otolaryngology, the American Medical Association and the County and State Medical Societies.

One son, Dr. M. W. Kneedler, survives him.

Dr. William Walter Lewis

Dr. William Walter Lewis was born on June 9, 1876, in St. Paul, Minnesota, and died there on July 27, 1935.

Receiving his medical degree from the University of Minnesota, Dr. Lewis began the practice of medicine in 1903 in St. Paul. Later, in 1911-1913, he took postgraduate courses abroad in eye, ear, nose and throat work at the University of Wien, Austria, and in Berlin, and returned to practice these specialties.

He was Assistant Professor of the Medical School of the University of Minnesota and on the staff of the Ancker Hospital for many years. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the American College of Surgeons, the Ramsey County Medical Society and the Minnesota Academy of Ophthalmology and Otolaryngology, having been honored with the presidency of the latter in 1926.

Dr. Lewis served as captain in the Medical Corps, U. S. Army, during the World War.

He was a member of the Presbyterian Church and a 32° Mason.

His widow, one daughter and one son survive him.

Dr. Clarence Loeb

Dr. Clarence Loeb was born on March 21, 1876, in Columbia, Missouri, and died on May 26, 1935, in Hubbard Woods, Illinois.

He was a graduate of the University of Missouri and the Marion-Sims College of Medicine in St. Louis, following which he was an interne at the St. Louis City Hospital, and began the practice of medicine in 1900 in St. Louis. Later he took special postgraduate work in Berlin and Vienna.

Dr. Loeb was Associate Professor of Ophthalmology at St. Louis University, Oculist to St. Louis City Hospital, the Alexian Brothers Hospital and the Mt. St. Rose Sanitarium, during his residence in St. Louis. At this time, too, he helped to establish the Missouri Association for the Blind, and was a member of the first executive committee. He served as first lieutenant in the M.R.C. Mobilization Camp, Nevada, Missouri, in 1916.

For many years Dr. Loeb was editor of the Transactions of the American Academy of Ophthalmology and Otolaryngology.

Dr. Lewis E. Maire

Dr. Lewis E. Maire, who died on January 9, 1935, at the age of seventy-nine, was graduated in 1881 from the Detroit Medical College and practiced in Grosse Pointe, Michigan.

He was a member of the American Academy of Ophthalmology and Otolaryngology, member of the Michigan State Medical Society and a past president of the Wayne County Medical Society.

Dr. Maire was interested in civic affairs and was formerly a member of the Board of Education and the Board of Health.

Dr. Alexander Dunbar McKelvey

Dr. Alexander Dunbar McKelvey was born in Brussels, Ontario, on September 17, 1885, and died in Toronto, Ontario, on January 6, 1935.

Receiving his preliminary education at Mount Forest High School, he took his medical degrees from the University of Toronto and spent many years in postgraduate work in the Massachusetts Eye and Ear Infirmary, Massachusetts General Hospital, and in Vienna, Basle and Berlin.

Dr. McKelvey held hospital and teaching appointments in the Toronto General Hospital and University of Toronto, and was a member of the American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, College of Physicians and Surgeons of Ontario, Massachusetts Medical Society, Ontario Medical Association, Academy of Medicine, Toronto, New England Ophthalmological and Otolaryngological Society.

He is survived by his wife, three children, and one sister.

Dr. Allyn Bernard Moise

Dr. Allyn Bernard Moise was born July 29, 1877, in Natchitoches, Louisiana, and died December 23, 1934, in Shreveport, Louisiana.

Receiving his early education at the Jesuit College and his medical degree from Tulane University in New Orleans, and taking postgraduate work in the Ear, Nose and Throat Senses Hospital, Dr. Moise took up the practice of his ear, nose and throat specialty in Shreveport.

He was former Assistant House Surgeon to Touro Infirmary of New Orleans, Acting Assistant Surgeon in the Public Health and Marine Hospital Service of the United States, and at the time of his death was on the staffs of the Schumpert Memorial Hospital, North Louisiana Hospital, Louisiana State Sanitarium and the Highland Sanitarium.

He was a member of the Shreveport Charity Board and Rotary Club, and a Fellow of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the American College of Surgeons, the Shreveport Medical Society, Louisiana State Medical Society, Southern Medical Association and the Tri-State Medical Society.

Dr. Moise is survived by his widow, two sons and one daughter.

Dr. William Valentine Mullen

Dr. William Valentine Mullin, for many years an honored Secretary of Instruction in the American Academy of Ophthalmology and Otolaryngology, and indefatigable in every possible voluntary and required service for this august body, was born in Iowa City, Iowa, on February 14, 1884, and died in Cleveland, Ohio, on April 25, 1935.

He attended the State University of Iowa and finished his medical studies at Gross Medical College of the University of Denver, graduating in 1908. He practiced general medicine in Southern Colorado for several years, taking up his specialty of ear, nose and throat work in Colorado Springs in 1912, with postgraduate work in otolaryngology in Berlin, 1914.

In 1926 he became head of the Department of Otolaryngology of the Cleveland Clinic.

Dr. Mullin was past Secretary and Chairman of the Section on Laryngology, Rhinology and Otology of the American Medical Association; Vice President and Treasurer, at one time, of the American Laryngological Association, and Secretary at the time of his death; a member of the American Board of Examiners in Otolaryngology; member of the American Laryngological, Rhinological and Otological Society, and the American Otological Society; Fellow of the American College of Surgeons; served during the World War, and on the editorial board of the Annals of Otology, Rhinology and Laryngology.

He is survived by his wife and one daughter.

It is difficult to adequately express in words our admiration and esteem for the magnificent service of this beloved friend, William Valentine Mullin. He seemed omnipresent in the great service of otolaryngology, covered an almost impossible field of work, held master keynote positions in many organizations, and yet with it all combined an ability of concentration in medical routine and intricate research which seems almost amazing in one so busy; add to those characteristics the rare and pleasing qualities of genial fellowship, with a greeting smile and handclasp which meant much to every member of this organization. He seemed on his feet all the time at the meetings, fraternizing with the members, and by his magnetic presence stimulating them to better work.

He was gracious in social life, a delightful house guest, keenly interested in athletics, particularly golf, fishing and baseball, enjoyed music and the theatre, but never let these interfere with his marvelous otolaryngological field of work.

We quote this expression that "perhaps his greatest attribute was his enjoyment in making friends and having many friends." None of us will ever forget "Bill" Mullin.

Dr. Clarence Anson Neighbors

Dr. Clarence Anson Neighbors was born in McPherson, Kansas, August 23, 1880, and died in Anaheim, California, on November 14, 1934.

He received his early education at the McPherson Academy and Ottawa University, and obtained his medical degree from Kansas City Medical College in 1905. He began his medical practice in Emporia, Kansas, but moved to Anaheim, California, in 1927, because of his health, and took up the practice of his specialty there.

Dr. Neighbors was a member of the American Academy of Ophthalmology and Otolaryngology, and past president of the Lyon County Medical Association.

In addition to his medical interests Dr. Neighbors was a trustee of the Baptist College of Kansas.

He is survived by his widow and three children.

Dr. Harry Edward Oesterling

Dr. Harry Edward Oesterling, of Winter Park, Florida, who died May 21, 1935, at the age of sixty-two, received his early education at Lindsley Academy, Wheeling, West Virginia, and graduated with his medical degree from the University of Pennsylvania in 1897, following which he did postgraduate work at Jefferson and Wills Eye Hospitals in Philadelphia, the New York Eye and Ear Infirmary, in Vienna, and at the Royal Hospital, Munich.

He began the practice of medicine in 1899 in Pittsburgh, later moving to Wheeling, West Virginia, and taking up eye, ear, nose and throat work.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, member of the West Virginia State Medical Association, and formerly on the staff of the Ohio Valley General Hospital of Wheeling.

In addition to his medical affiliations, Dr. Oesterling was a trustee of Rollins College, Winter Park, Florida, the Fort Henry Club of Wheeling, West Virginia, and Vice President of the Library Board of Winter Park, Florida.

Dr. Oesterling was a Presbyterian and a Mason.

He is survived by his widow.

Dr. William Nelson Offutt

Dr. William Nelson Offutt, who died in Lexington, Kentucky, on April 3, 1935, at the age of fifty-eight, was educated in private schools and Georgetown College at Georgetown, Kentucky, and received his medical degree from the Hospital College of Medicine in Louisville in 1902. He began the practice of medicine in 1903 in Lexington, after postgraduate work in his specialty in London.

At the time of his death Dr. Offutt was on the staffs of the Good Samaritan and St. Joseph's Hospitals. He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, member and past president of the Eye, Ear, Nose and Throat Section of the Kentucky State Medical Society, and a member of the Southern Medical Association.

Dr. Offutt was an elder of the Second Presbyterian Church at Lexington, and was also keenly interested in farming and stock breeding, Boy Scout activities, hunting and fishing.

He is survived by his widow, one son, a physician, three daughters and one brother.

Dr. Victor Ray, Sr.

Dr. Victor Ray, Sr., was born in New York City on June 28, 1866, and died in Cincinnati, Ohio, on October 25, 1934.

The first sixteen years of his life were spent abroad. Returning to the United States he entered the Massachusetts Institute of Technology, graduating in 1887, and took the position of chemist with the Anaconda Copper Company, later becoming manager and general superintendent of that company. He had, however, desired to study medicine and after nine years in the engineering field, he entered the Miami Medical School, Cincinnati, in 1894, receiving his medical degree in 1897. Following his interneship in the old City Hospital, he began the practice of ophthalmology with his brother-in-law, Dr. Robert Sattler.

In 1926, Dr. Ray was appointed Professor of Ophthalmology in the Medical Department of the University of Cincinnati, having previously served as Assistant Professor. In 1926, he was elected President of the Academy of Medicine. At the time of his death Dr. Ray was a member of the staff of the Good Samaritan Hospital and of the Ophthalmic Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology and of the American Medical Association.

Dr. Ray was an expert in ophthalmic surgery, a diagnostician of excellent judgment and wide clinical experience, and possessed of great personal charm.

A wife, two daughters, and one son, Dr. Victor Ray, Jr., survive him.

Dr. William Scott Renner

Dr. W. Scott Renner was born at Jordan Station, Ontario, on March 31, 1861, and died in Buffalo, New York, on October 10, 1934.

Receiving his early education at St. Catherine's Collegiate Institute, St. Catherines, Ontario, he graduated from McGill

University of Montreal in 1884 with the degree of M.D., C.M., following which he took postgraduate work in Vienna and Berlin. Returning he took up the practice of his specialty of nose, throat and ear in Buffalo in 1885. Dr. Renner was for several years Professor of Laryngology at Niagara University, and Clinical Professor of Laryngology and Associate Professor of Otolaryngology at the University of Buffalo. He was at various times on the staffs of the Buffalo Eye and Ear Infirmary, Buffalo Children's Hospital, Sisters of Charity Hospital, Deaconess Hospital, Emergency Hospital, Mercy Hospital, Memorial Hospital, Buffalo City Hospital and the Erie County Hospital, Buffalo, and the J. N. Adam Hospital, Perrysburg, N. Y.; consulting specialist in diseases of the ear, nose and throat, U. S. Public Health Service and of the U. S. Veterans' Bureau.

Dr. Renner was a member of the American Academy of Ophthalmology and Otolaryngology, American Laryngological Association, American Laryngological, Rhinological and Otological Society and American Otological Society; Fellow of the American College of Surgeons; past president of the Buffalo Academy of Medicine; member of State and County Medical Societies and of the American Medical Association.

Dr. Renner was the author of various articles dealing with diseases of the nose, throat and ear, a Republican, an Episcopalian, and a member of Nu Sigma Nu Fraternity.

Dr. Duncan Campbell Smyth

Dr. Duncan Campbell Smyth was born on May 8, 1885, at Port Hood, Nova Scotia, and died June 11, 1935, in Brookline, Massachusetts.

His early education was obtained at the Port Hood Academy and later at the University of St. Francis Xavier's College in Antigonish, N. S. Receiving his medical degree from Harvard Medical School in 1909, he interned at the Massachusetts Eye and Ear Infirmary, and began to practice medicine in Boston.

For many years Dr. Smyth was an instructor in Laryngology at the Harvard Medical School and at the time of his death was on the staffs of the Symmes Arlington Hospital, and the Massachusetts General Hospital, and Senior Surgeon in the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Laryngological Association, the American Laryngological, Rhinological and Otological Society, the American Otological Society, the New England Otological and Laryngological Society, and in 1933 was Vice President of the American Bronchoscopic Society.

Dr. Smyth invented two bronchoscopic instruments and one operation bears his name. He was a consistent contributor of medical articles.

In addition to his medical affiliations, Dr. Smyth was keenly interested in birds and dogs, Websteriana, and civic affairs.

He is survived by his widow, a son and a daughter.

Dr. Morie Frederick Weymann

Dr. Morie Frederick Weymann was born in St. Joseph, Missouri, in 1899, the son of a physician, and died in Los Angeles, California, on January 13, 1935.

He was educated at the University of Missouri and Washington University School of Medicine, from which he graduated in 1922, one of the outstanding men of his class. In 1924 he moved to Los Angeles and began the practice of ophthalmology, becoming Clinical Professor of Surgery (ophthalmology) at the Southern California Medical School, and on the staff of St. Vincent's Hospital.

Dr. Weymann joined the editorial board of the American Journal of Ophthalmology in 1932, and contributed much to the Journal.

He was a member of the American Academy of Ophthalmology and Otolaryngology, holding the office of Third Vice-President at the time of his death, the American Ophthalmological Society, and the Pacific Coast Ophthalmological Society.

He is survived by his widow.

Dr. Douglas Fox Wood

Dr. Douglas Fox Wood was born December 10, 1874, in Faribault, Minnesota, and died on February 9, 1935, at St. Petersburg, Florida.

Following his early education at the Shattuck Military School in Faribault, and obtaining his medical degree at McGill Uni-

versity in Montreal, in 1900, Dr. Wood began the practice of medicine in Hanska, Minnesota. Later he did postgraduate work in eye, ear, nose and throat in Chicago and New York, and continued his studies at the University of Vienna. He then took up the practice of his specialties in Minneapolis in 1909.

For many years Dr. Wood was on the staffs of the Northwestern, Abbott, and Swedish Hospitals, and was very active in work for the blind in his city and state. He was instrumental in organizing the first school eye clinic and the sight-saving classes of the public schools of Minneapolis.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, member of the Minnesota State Medical Association, former Vice President of the Hennepin County Medical Society and on their Board of Censors, and Past President of the Minnesota Academy of Ophthalmology, Otology and Laryngology.

In addition to his medical interests, Dr. Wood was Past President of the State Society of Sons of the American Revolution and first President of the Minneapolis Chapter, and a Mason.

He is survived by his widow and one daughter.

Dr. John Westley Wright

Dr. John Westley Wright, of Columbus, Ohio, who died on May 23, 1935, at the age of ninety-two, was graduated from the Cincinnati College of Medicine and Surgery with his medical degree in 1873.

He was a member of the American Academy of Ophthalmology and Otolaryngology. At one time Dr. Wright was on the staff of the Protestant Hospital, now known as the White Cross Hospital, from 1891-1910 was Professor of Ophthalmology at Ohio Medical University and Starling-Ohio Medical College and was later Emeritus Professor of Clinical Ophthalmology at the Ohio State University College of Medicine.

Dr. Wright was also a Civil War veteran, and is known for his authorship of a "Textbook on Ophthalmology."

Dr. Henry Byrd Young

Dr. Henry Byrd Young was born at Monmouth, Illinois, on March 20, 1851, and died at Burlington, Iowa, on December 10, 1934.

Graduating from Chicago Medical College (later affiliated with Northwestern University) in 1875, he entered practice in Southern Minnesota. Later he went to Vienna for advanced study in ophthalmology and otology, and in 1879 began the practice of these specialties in Burlington, and remained there fifty-five years.

At various times Dr. Young was on the staffs of St. Francis, Burlington, Protestant and Mercy Hospitals. He was a life member of the American Academy of Ophthalmology and Otolaryngology, past President of the Iowa State Medical Society, an honorary member of the Chicago Ophthalmological Society, and had served three terms as President of the Des Moines County Society — the last term in 1931.

For many years he was Consulting Ophthalmologist to the Burlington Railroad and was on the draft board all through the World War.

Dr. Young had traveled extensively, was keenly interested in life, and was the author of many papers in his chosen field in medicine.

He is survived by his daughter, a niece and three grandchildren.

Dr. Edward Frank Ziegelman

Dr. Edward Frank Ziegelman was born in La Crosse, Wisconsin, on July 10, 1884, and died October 12, 1934, on board a Chicago & Northwestern train between Maywood and Chicago.

Dr. Ziegelman was educated at the University of Wisconsin, Oregon Medical College, graduated from Jefferson Medical College with the degree of M.D., in 1912, and served as interne in the Episcopal Hospital, Philadelphia and Kings County Hospital, Brooklyn, New York.

Following World War service with the United States Army abroad as General Surgeon, Dr. Ziegelman practiced in Portland, Oregon, until September, 1924, leaving to take up postgraduate work in ear, nose and throat at Stanford Medical School, Berlin, London, Paris and Vienna; then returned to practice his specialty in San Mateo and San Francisco, California.

Dr. Ziegelman was a member of the American Academy of Ophthalmology and Otolarynogolgy, the American Laryngological, Rhinological and Otological Society, the American Otological

Society, diplomate of the American Board of Otolaryngology, California Medical Association, Pacific Coast Oto-Ophthalmological Society, a Fellow of the American College of Surgeons, and a life member of the American Medical Association of Vienna. At the time of his death he was Assistant in the Department of Otology, Rhinology and Laryngology of Stanford University and Medical School, Instructor in Anatomy of the College of Physicians and Surgeons, Dental Department, San Francisco, California; was a member of the staffs of St. Vincent's Hospital and Multnomah Hospital in Portland, Oregon, and Oto-Rhino-Laryngologist to the Mills Memorial Hospital and the San Mateo Community Hospital, both of San Mateo, California.

Dr. Ziegelman was the author of many valuable scientific articles.

He is survived by his mother and two sisters.

REPORT OF THE AMERICAN BOARD OF OPHTHALMOLOGY

Since the last meeting of the Academy in Chicago, 1934, the Board has conducted three examinations:

San Antonio	November 12, 1934
Philadelphia	June 8, 1935
New York	June 10, 1935

Since the first examination held in December, 1916, we have conducted 48 examinations and a total of 1,248 certificates have been issued.

This is the first year that the Board has functioned with a membership of twelve instead of nine. As a result fewer outside Associate Examiners have been called on. The need for Associates has been further reduced by conducting examinations throughout the full day, instead of only halfday as formerly.

The new Directory was issued this year and 1,240 copies were sent gratis to diplomates, hospitals, medical schools and other institutions.

Three syllabuses have been published covering the subjects of Refraction, Pathology, and of Optics and Visual Physiology. It is hoped that these will be of service not only to the candidate

by making more clear how he is to prepare himself for the examination, but also to teachers in arranging courses on these subjects. Several other syllabuses are now being drafted and will be ready shortly. There has been a great demand for these by candidates and by the heads of the departments in various teaching institutions.

PERSONNEL OF THE BOARD

Term Ending	*A.M.A.*	*A.O.S.*	*Academy*
Dec. 31, 1935	Dr. Lancaster	Dr. McCool	Dr. Thos. D. Allen
Dec. 31, 1936	Dr. John Green	Dr. L. C. Peter	Dr. S. Judd Beach
Dec. 31, 1937	Dr. Ellett	Dr. Evans	Dr. W. H. Wilder
Dec. 31, 1938	Dr. Crisp	Dr. Berens	Dr. E. L. Goar

(Report accepted with thanks.)

REPORT OF THE AMERICAN BOARD OF OTOLARYNGOLOGY

DR. W. P. WHERRY: The American Board of Otolaryngology, as usual, has held two examinations during the current year. In New York, in June, approximately 150 candidates were examined, and 64 were examined at Cincinnati. I am sorry that I had not prepared a written report for you, but I can assure you that progress has been made and that the boys are all working hard.

(The report was accepted with thanks and placed on file.)

Reports of Standing Committees

REPORT OF THE AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

1. Our Committee has recommended that visual acuity of 20/20 in each eye, without correcting lenses, be required as the minimum visual standard for admission to the United States Military Academy.

2. We approved the suggestion that all advertising matter concerning optical apparatus, including lenses, be passed upon by competent individuals before publication in the American Ophthalmic Journals. This Committee announced its willingness to cooperate in this work.

3. The syllabus on Optics and Visual Physiology adopted by the American Board of Ophthalmology was approved as the minimum requirement for preparation in the field of Physiologic Optics. Copies may be obtained from the secretary of the American Board of Ophthalmology.

4. We believe that serious consideration should be given to the simplification and cost reduction of ophthalmic equipment.

5. Among the activities of this committee which are now in progress are:

A. Studies of visual acuity are being conducted by us in the Department of Psychology, University of Michigan.

B. The study of the lensometer from the standpoint of practicality and cost.

C. The establishment of a bureau where information may be obtained by American ophthalmologists about ophthalmic equipment manufactured anywhere in the world.

D. The study of foreign instruments which seem to have outstanding merit.

E. The following foreign instruments have been found useful in the treatment of heterophoria and heterotropia: the Synoptiscope, the Synoptophore, and the Orthoptoscope, all of English manufacture.

The English Fincham Coincidence Optometer, the Dobson Concentrator, and the German Bush Combined Tonometer and Microscope are among the instruments which seem worthy of further study.

Respectfully submitted,

CONRAD BERENS, M.D., *secretary*

Members of the Committee:
EDWARD JACKSON, M.D., *chairman*
CHARLES A. BAHN, M.D.
S. JUDD BEACH, M.D.
WILLIAM L. BENEDICT, M.D.
ALFRED COWAN, M.D.
JOHN N. EVANS, M.D.
WALTER B. LANCASTER, M.D.
WILLIAM H. LUEDDE, M.D.

(The report was received with thanks.)

EXTRAMURAL GRADUATE INSTRUCTION

DR. HARRY S. GRADLE: Mr. President, the Committee on Extramural Graduate Instruction, for the past year, has been extremely busy doing nothing. Various courses throughout the country have been given without the aid or assistance of the Committee on Extramural Instruction. They have seemed to regard us as useless as the policeman at the red-light crossing. Consequently, the Committee has no progress to report and respectfully begs to be discharged.

I might amplify that a little. The Committee was formed with the idea of aiding the various institutions around the country that are giving courses to eye, ear, nose and throat men. This Committee spent a good deal of time in preparing a list of acceptable teachers, the time that they would be available, their locality, and so forth and so on. We sent word around the country and we have communicated with a large number of the institutions that are providing courses, and have been more or less politely informed that these institutions were capable of conducting their own courses without the aid of this Committee. Consequently, the Committee feels that they are more or less unnecessary and that the time evidently is not ripe for a coordinating committee for extramural instruction.

PRESIDENT EAGLETON: I think the report of the Committee on Extramural Graduate Instruction is quite characteristic of the Academy. This group of men has devoted a tremendous amount of time to the work and the opportunities they have offered have not been taken. They do not want to be carried on simply because they are a committee. I think they should be discharged with thanks. We are sorry that the American institutions did not avail themselves of the opportunities offered by the Academy.

(Upon motion made by Dr. Burch, the Committee was discharged with thanks.)

NATIONAL OTOLARYNGOLOGICAL LIBRARY

DR. RALPH A. FENTON: Mr. President, May I be permitted to say, for Dr. Shurly, that without knowing when this report would be called for, the Public Relations Committee arranged a broadcast for him at this hour.

I would say Dr. Shurly's report is embodied in his recommendations to the Teachers' Section meeting the other night, and in that form will reach the membership of the Academy.

PREVENTION AND AMELIORATION OF DEAFNESS

DR. HORACE NEWHART: Mr. President, and Fellows of the Academy: Your Committee has prepared and had printed a rather lengthy report which has been placed in the hands of our President and Secretary and to which others are welcome at the close of the meeting. Therefore, we will, very briefly, summarize in a few words some of the points in this report.

During the past year the members have carried on very actively — more actively than ever before — along the lines laid out for us, with which you are all familiar.

Outstanding in this work, we would mention a study of the teaching of otology to undergraduates in our medical schools, because we have found that one of the greatest obstacles to progress in different communities in the work for the hard of hearing and on the question of deafness has been the lack of specific knowledge on the part of the medical men regarding the needs of this group and the possibilities. Therefore, we recommend that the courses in many of our schools be revised and in some cases expanded to include the teaching of these subjects.

Last year, stimulated largely by the generous grant of our Council for funds, we were able to set up a research project, a combination of effort on the part of the School of Electrical Engineering and the Division of Otolaryngology of the Medical School of the University of Minnesota, its specific purpose being to study the efficiency of hearing aids, and also we have just taken on the study of audiometry, in which there is a great deal of interest.

So far, we are able to report there is such a variation in the efficiency of carbon microphone hearing aids as now produced, that it is necessary, in order to be fair to our patient, to recommend that he try out several instruments of different makes before making a choice. In this matter the otologists can be of great help.

To discourage an undesirable practice that has come to the notice of this particular Committee in that the hearing agents have been known to offer — I don't want to use the word "bribes" — bonuses to individuals for referring to them prospects for purchasing their instruments, we introduced before the American Otological Society and the American Laryngological, Rhinological and Otological Society, at their last annual meetings in June, this resolution:

"WHEREAS, Certain distributors and agents of electric hearing devices have offered to physicians and others a bonus or commission for referring to them hard of hearing persons, to whom they effect a sale,

"BE IT RESOLVED, By the.....
that it condemns as unethical and unfair to the purchaser, the practice on the part of any agent or distributor of a hearing device who offers to pay to any physician a bonus or commission for referring a purchaser of such device. The acceptance of such a bonus or commission by a practicing physician is interpreted as a violation of the principle of ethical practice."

This resolution was unanimously adopted by both organizations and I think is under consideration in our Council subject to the approval of the American Academy of Ophthalmology and Otolaryngology.

A feature in which we have been greatly interested in our work has been the exhibits held at the American Medical Association meeting, at the Minnesota State Medical Association and at our meeting this week. Your Committee feels that there is need for us to know more about the work in this line, and the response which we have had to inquiries, not only among otolaryngologists, but pediatricians, neurologists, and others, has been most gratifying, and we feel the efforts are very much worth while.

We have also carried on a study of the hearing loss among rural school children as compared with city school children, and we find a great need of larger effort with the country school children. Also, as a side issue, we have confirmed the often made statement that the hearing tests made by public school nurses are absolutely inadequate. The discrepancy is so great it is appalling.

We recommend the putting forth of greater efforts by the medical profession to secure the adoption of accurate hearing

tests of all school children as a part of every school health program and that a careful test of the hearing acuity be included in the periodic, routine, health examination of individuals of all ages.

We would recommend, further, the formation of similar committees of like purpose by medical societies in our different states and larger cities, to be composed of men interested in the problem of better hearing. The well-defined activities of such committees would soon effect a definite reduction in the large number of our population afflicted with a handicapping hearing loss; would materially increase the amount of medical work for the individual physician and would be a real factor in demonstrating to the laity the public-mindedness of our profession.

Your Committee wishes to expand its membership to fill in the gaps caused by the lamented death of Dr. Ziegelman and the resignation of Dr. Tobey, by the appointment of Dr. Furstenberg and Dr. Marshall Taylor as members.

Respectfully submitted,

JOHN F. BARNHILL
GORDON BERRY
THOMAS E. CARMODY
E. H. CARY
GEORGE M. COATES
HOWARD V. DUTROW
AUSTIN A. HAYDEN
FREDERICK T. HILL
BURT R. SHURLY
CLAUDE T. UREN
HORACE NEWHART, *chairman*

(Upon motion made by Dr. Fenton, the report was received with thanks.)

PUBLIC RELATIONS

Continuing the policy of public instruction heretofore authorized by the Council, your Committee on Public Relations secured abstracts and prepared thirty press releases and three broadcasts before reaching Cincinnati. The services of a press agent approved by the local medical society were secured for the distribu-

tion of these releases; and a considerable number of other releases from papers received late and covering current happenings of the session were prepared here, and special interviews were supervised by Committee members. Particular interest in the events of the session was, as last year, manifested by national periodicals and services reporting on scientific medicine.

As evidenced by clippings submitted to the Council, better control of lay interpretation has been secured at this session and in Chicago than heretofore.

In accordance with the rules of the American Medical Association, names of all local physicians were excluded from all news stories except the preliminary announcement, and your committee wishes to express its appreciation for the hearty cooperation of Drs. Iglauer and King, as well as the editors and special writers for national and local press associations, local newspapers, and periodicals reporting on scientific medicine.

In addition to broadcasts by Dr. Burch, on "The Academy and its Scope"; Dr. Davis, on "Crossed Eyes," and Dr. Shurly, on "Ears and Sinuses," the chairman of your Committee gave a radio interview preceding the Academy meeting last week, covering the anniversary session and paying tribute to Drs. Vail and Holmes; Dr. Brunner gave a radio interview on "Socialized Medicine in Austria," and Dr. McKee prepared a national broadcast on "The Use of Glasses."

Continuation of current policies has been recommended to the Council by your Committee.

Respectfully submitted,

RALPH A. FENTON, *chairman*
JOHN J. SHEA
ERLING W. HANSEN

PRESIDENT EAGLETON: The work of this Committee is probably further reaching than any other activity of this Society that is not truly medical.

One of the disgraces of the medical profession — and there are very few — has been the amount of personal exploitation that has gone on in the public press by certain members who, seeing

an opportunity to increase their practices, have used their position on the programs of different medical societies.

The Council gave a great deal of attention, under the advice of Dr. Wherry and Dr. Fenton, some years ago, to this matter, with the result that the condition during the last two years has wonderfully improved, increasing the dignity and the influence of the profession and making those men who would exploit themselves feel that not only is it a wrong thing but it is a dangerous thing. Personally, I want to thank Dr. Fenton for what he has done.

(The report was accepted with thanks.)

OPHTHALMIC PATHOLOGY

Dr. Harry S. Gradle: There is very little to report in addition to what has been reported in previous years. The Army Medical Museum is carrying on the Registry of Ophthalmology, as it has for the past fifteen years. It is continuing the work. There are continuous specimens coming into the museum which are being handled in the same beautiful manner in which they have been handled in the past. The museum now numbers over 5,000 specimens and bids fair to be one of the great ophthalmic collections of all time.

It is urged that all members continue to send interesting material to the museum as they have in the past. The scope of the loan sets, which were manufactured by the museum with Academy money, is increasing continuously and these sets are in almost constant circulation. They are being revised by the present curator of the museum and the histories and descriptions are being more or less rewritten to conform to the sets that are being put out by Dr. Theobald for the American Board of Ophthalmology.

The work at the museum is continuing and deserves the further support of the Academy.

(The report was accepted with thanks.)

REPORT OF THE SENIOR MEMBER OF THE COUNCIL

Through the cooperation of the Army Medical Museum, there has been created a Registry of Otolaryngologic Pathology. Members are urged to contributed interesting material.

Committee appointed: Dr. Joseph C. Beck, Chicago, Illinois.

The Board of Secretaries has been authorized to appoint assistant secretaries to create a group capable of taking over the duties of a Secretary.

The Finance Committee reports the official audit of the 1934 budget expenses as correct.

That the capital amount investment be confined to United States and Canadian Government bonds.

That a reliable finance company be suggested to analyze the investment portfolio.

The following research appropriations are authorized:

Dr. William J. McNally	$ 750.00
Dr. Ralph A. Fenton	250.00
Dr. Horace Newhart for the Committee on the Hard of Hearing	250.00
Dr. M. H. Lurie	200.00
Army Medical Museum	1000.00
International Committee on Trachoma	100.00

The Activities Committee has advised no change in the convention setup of 1936. Approved by the Council.

The list of those delinquent in dues was carefully analyzed and the Executive Secretary was authorized to drop from the rolls on January first of each year those delinquent for four years or more.

A proposal was submitted that members either dropping out of their local eye, ear, nose and throat societies, or not at present a member of such local societies, be dropped from Academy membership.

The Council deemed it wise at the present time to oppose such a suggestion, which, if put in force, obviously would necessitate a reorganization of the structure of the Academy.

A proposal was offered to create a Bibliographic Bureau to which members could send requests for reference lists on selected subjects. That this service be rendered at a low minimum cost.

The Council instructed the Secretaries to investigate and offer recommendations.

The minutes of the Teachers' Section to be printed in the Fall Bulletin and made easily available to teaching institutions.

The Transactions of 1935 are to be dedicated to Doctor Allen Greenwood of Boston, Massachusetts.

The Extramural Graduate Committee to be dropped.

The Public Relations Committee to be continued as at present.

The Council approved the granting of a membership certificate to new members. That such a certificate be made available to old members at one dollar.

To encourage the development of suitable moving picture films of an educational type, the Council set aside a sum of money to be expended under the direction of the Board of Secretaries.

(The report was accepted as read.)

New Business

DR. FRANK E. BURCH: I move that the Committee on the National Otolaryngological Library be discharged and the services of that Committee be incorporated in the Surgeon General's Library.

(The motion was seconded by Dr. Ralph Fenton, put to a vote and carried.)

Election of Members

DR. W. P. WHERRY: The Council has instructed me to advise that the following have been elected to membership by the Council:

Anderson, William Banks, McPherson Hospital, Durham, N. C., Op.
Barnes, Lancelot, 170 St. George St., Toronto, Ontario, Op.
Baron, Shirley Harold, 309 State Street, New London, Conn., ALR
Beck, Robert J., 105 Main St., Saranac Lake, N. Y., Op.
Bellows, John, 58 E. Washington St., Chicago, Ill., Op.
Berman, Philip G., 174 Central St., Lowell, Mass., Op.
Berns, Charles, 303 Osborn Bldg., Cleveland, Ohio, ALR
Best, Lynwood Kent, 151 Rock St., Fall River, Mass., Op.
Bilchick, Edwin B., 470 Park Ave., New York, N. Y., ALR
Billings, Robert John, Jenkins Arcade, Pittsburgh, Pa., Op.
Boice, Ralph Edwin, 230 Michigan St., Toledo, Ohio, Op.
Bradley, Basil Vincent, Medical Arts Bldg., Toronto, Ontario, ALR
Bristow, Walter James, 1512 Marion St., Columbia, S. C., ALR
Browning, Andrew Johnson, 624 Medical Arts Bldg., Portland, Oregon, Op.
Browning, Birt Lee, 208 Grand St., Newburgh, N. Y., ALR
Bruno, Joseph M. L., 31 Jefferson St., Brooklyn, N. Y., Op.
Buffington, Wiley Ross, 1206 Hibernia Bank, New Orleans, La., Op.
Bunnell, Chester Wm., 121 E. 60th St., New York, N. Y., ALR
Burke, B. Russell, 478 Peachtree St., Atlanta, Ga., ALR
Burman, Herman Joseph, 1235 Grand Concourse, New York, N. Y., ALR
Busler, Howard Sherwood, N. E. Cor. Baltimore and Owen Aves., Lansdowne, Pa., ALR
Cecha, Albin Hastings, Naval Hospital, Quantico, Virginia, Op.
Cohen, Nathan Bertram, 232 State St., Perth Amboy, N. J., ALR
Colgin, William Edward, 605 Columbus Ave., Waco, Texas, Op.
Combacker, Leon C., 101½ Lincoln Ave., W. Fergus Falls, Minn., Op.
Conboy, John Edward, 2257 University Ave., New York, N. Y., Op.
Conway, William S., 314½ Howard St., Petoskey, Mich., ALR
Coon, Eugene H., 131 Fulton Ave., Hempstead, N. Y.
Courtney, Robert Hull, 501 E. Franklin St., Richmond, Va., Op.

Cracovaner, Arthur J., 103 E. 78th St., New York, N. Y., ALR
Crage, Francis M., 30 N. Michigan Ave., Chicago, Ill., Op.
Croushore, James Elliott, 2501 W. Grand Blvd., Detroit, Mich., ALR
Dickerson, William Edwin, 802 Masonic Temple, Danville, Va., ALR
Dirion, Josephine Koob, 835 Rose Bldg., Cleveland, Ohio, Op.
Dunaway, Carl Ellis, 1209 Huntington Bldg., Miami, Fla., ALR
Edwards, Stephen Austin, Home Guard Bldg., Van Wert, Ohio, ALR
Faier, Samuel Z., Medical Arts Bldg., Omaha, Nebr., ALR
Farrell, James Irving, 250 Genesee St., Utica, N. Y., Op.
Feldman, Louis A., 259 New York Ave., Brooklyn, N. Y., Op.
Fellows, Manley Fording, 815 Medical Arts Bldg., Duluth, Minn., Op.
Ferguson, Everett Russell, P. & S. Hospital, Cleveland, Tenn.
Fernandez, Francisco M., 343 Ingraham Bldg., Miami, Fla., Op.
Fishman, Louis Zolo, 1439 Summerdale Ave., Chicago, Ill., ALR
Foster, Harold, 9205—35th Ave., Jackson Heights, New York, N. Y., ALR
Foster, Warren Henry, 408—217 Fifth Avenue, S., Clinton, Iowa, ALR
Foster, Wayne Jones, 601 Higley Bldg., Cedar Rapids, Iowa, ALR
Furlong, Thomas Francis, Jr., 601-2 Times Medical Bldg., Ardmore, Pa., ALR
Garrett, Ernest Walter, 952 Hanna Bldg., Cleveland, Ohio, ALR
Gemeroy, Joseph Conrad, Henry Ford Hospital, Detroit, Mich., ALR
Gibson, Glen Gregory, Beury Bldg., 3701 N. Broad St., Philadelphia, Pa., Op.
Goldberg, Sylvan S., 1917 Wilshire Blvd., Los Angeles, Calif., ALR
Goldthwaite, Ralph Harvard, Gorgas Hospital, Canal Zone, Panama, OALR
Gooch, John Barr, 1027 Canal Bank Bldg., New Orleans, La., ALR
Green, Hyman Sylvan, 1005 Belmont Avenue, Chicago, Ill., ALR
Griswold, George Wayne, 211 W. 3rd St., Roswell, N. M., ALR
Gundersen, Trygve, 5 Bay State Roard, Boston, Mass., Op.
Haire, Paul Gregory, 6 Pleasant St., Malden, Mass., Op.
Hammond, Arthur, 918 David Whitney Bldg., Detroit, Mich., ALR

Hand, Frank, 490 Post St., San Francisco, Calif., ALR
High, Clifton Earl, Small Bldg., Wellington, Texas, Op.
Hilgartner, Henry Louis, Jr., 209 Norwood Bldg., Austin, Texas, Op.
Hochfilzer, John J., 641 Lowry Medical Arts Bldg., St. Paul, Minn., ALR
Holmes, Will Wanee, Masonic Temple, Logansport, Ind., ALR
Hornback, George Allen, 500 Broadway, Hannibal, Mo., ALR
Horner, Warren Douglas, 384 Post St., San Francisco, Calif., Op.
Hull, Allen, 1847 Benson Ave., Brooklyn, N. Y., Op.
Jervey, James Wilkinson, 101 Church St., Greenville, S. C., OALR
Johnson, Henry Peter, 32 Deering St., Portland, Me., ALR
Johnson, John L., 33 Grove St., Bangor, Me., ALR
Jones, David H., 140 E. 54th St., New York, N. Y., LR
Judd, Delbert Kenneth, 1020 Medical Arts Bldg., Omaha, Nebr., ALR
Kaplan, Samuel, 1 Nevins St., Brooklyn, N. Y., ALR
Kapp, Carl Gailard, 440 Westminster Ave., Elizabeth, N. J., ALR
Kilgore, George L., Bank of America Bldg., San Diego, Calif., Op.
Kirkbright, James Calvert, 40 E. 61st St., New York, N. Y., ALR
Kully, Herman Edward, 1316 Medical Arts Bldg., Omaha, Nebr.
LaMotte, William Oscar, Medical Arts Bldg., Wilmington, Del., ALR
LaReau, Hector George, 4803 Lincoln Ave., Chicago, Ill., ALR
Lefevre, Willis Hess, 234 N. Duke St., Lancaster, Pa., ALR
Lemel, Samuel Louis, 1148 Euclid Ave., Cleveland, Ohio, ALR
Levitt, Jesse Marcus, 734 Ocean Ave., Brooklyn, N. Y., Op.
Lowenthal, Gerson, 40 E. Fourth St., Cincinnati, Ohio
MacDonald, Philip George, 115 E. 61st St., New York, N. Y., ALR
MacLean, Angus Lloyd, 1201 N. Calvert St., Baltimore, Md., Op.
Macnie, John Percival, 635 W. 165th St., New York, N. Y., Op.
Mann, Albert Howell, 209 Pine St., Texarkana, Arkansas-Texas, ALR
Marchman, Oscar Milton, 814 Medical Arts Bldg., Dallas, Texas, ALR
Marshall, Hal E., 227 E. Douglas St., Wichita, Kansas, ALR
Matusow, Abraham Perlin, 1100 Grand Concourse, New York, N. Y., ALR
Maxwell, Harvey Cecil, Fort Sam Houston, Texas, OALR
McLellan, Thomas George, First National Bank Bldg., Connellsville, Pa., Op.

McPherson, Samuel Dace, McPherson Hospital, Durham, N. C., ALR
Mesirow, Maurice Elias, 185 N. Wabash, Chicago, Ill., Op.
Miller, George Leslie, 14805 Detroit Ave., Lakewood, Ohio, Op.
Miniter, Francis Gabriel, 419 Boylston St., Boston, Mass., ALR
Monahan, James Joseph, 31 S. Jordin St., Shenandoah, Pa., ALR
Moore, J. Hallock, 1050 Fifth Ave., Huntington, W. Va., ALR
Mullen, Clifford John, 338 Brotherhood Bldg., Kansas City, Kansas, Op.
Nethercut, Glenway Warren, 25 E. Washington St., Chicago, Ill., Op.
Northington, Page, 20 E. 53rd St., New York, N. Y., ALR
Owsley, John Quincy, Bureau of Medicine and Surgery, Navy Department, Washington, D. C., ALR
Parry, Thayer L., Second National Bldg., Akron, Ohio, ALR
Parsell, Louis Alger, 755 Paulsen M. & D. Bldg., Spokane, Wash., ALR
Pfeiffer, Raymond Louis, 635 West 165th St., New York, N. Y., Op.
Pino, Ralph Harrison, 1001 David Whitney Bldg., Detroit, Mich., Op.
Pochert, Rolland C., 409 Matthews Bldg., Owosso, Mich., ALR
Porter, Whitney C., Republic Bldg., Denver, Colo., Op.
Potter, Jacob John, University Hospital, Iowa City, Iowa, ALR
Pressman, Joel Jay, 1917 Wilshire Blvd., Los Angeles, Calif., ALR
Quiring, Walter O., 101 First West, Hutchinson, Kansas, ALR
Reimers, Robert S., 613 Eighth St., Fort Madison, Iowa, ALR
Richman, Frances, 522 Ocean Ave., Brooklyn, N. Y., Op.
Riddell, Malcolm M., 112½ Front St., Fremont, Ohio, ALR
Rimer, Frank Hogan, 7026 Jenkins Arcade, Pittsburgh, Pa., ALR
Risch, Otto Carl, 104 E. 40th St., New York, N. Y., ALR
Rones, Benjamin, 1610 Eye Street, N.W., Washington, D. C., Op.
Row, Dunn Hamilton, 23 E. Ohio St., Indianapolis, Ind., Op.
Sachs, Benjamin, 483 Beacon St., Boston, Mass., Op.
Schechter, Solomon, 1749 Grand Concourse, New York, N. Y., ALR
Schwartz, Leo, Jr., 1 West 73rd St., New York, N. Y., ALR
Secoy, Harry Raymond, Everett Clinic, Everett, Wash., ALR
Selinger, Elias, 55 E. Washington St., Chicago, Ill., Op.
Semenov, Herman, 500 S. Lucas St., Los Angeles, Calif., ALR
Shipman, James Shelby, 542 Cooper St., Camden, N. J., Op.
Sparber, Herman, 4701 Fifteenth Ave., Brooklyn, N. Y., Op.

Steinbugler, William F. C., 515 Ocean Ave., Brooklyn, N. Y., Op.
Stern, Milton J., 1300 First National Bank, Lexington, Ky., Op.
Stone, Leslie Ogburn, U. S. S. New Orleans, care of Postmaster, San Pedro, Calif., ALR
Swing, Frank Ulmer, Doctors Bldg., Cincinnati, Ohio, ALR
Taylor, H. Marshall, 111 W. Adams St., Jacksonville, Fla., ALR
Thomas, W. Maxwell, 1228 Medical Arts Bldg., Dallas, Texas, Op.
Van Alyea, Oliver Edmond, 30 N. Michigan Ave., Chicago, Ill., ALR
Voorhees, Earle Winfred, 25 Market St., Poughkeepsie, N. Y., ALR
Walker, Orville Jackson, 308 Home Savings & Loan Bldg., Youngstown, Ohio, ALR
Walls, Edward Garland, 1230 Maison Blanche Bldg., New Orleans, La., ALR
Weinstein, Adolph Joseph, 244 W. 74th St., New York, N. Y., ALR
Wescott, Virgil, 30 N. Michigan Ave., Chicago, Ill., Op.
Wheeler, Maynard C., 30 W. 59th St., New York, N. Y., Op.
Wible, LeRoy Ellwood, 516-17 Empire Bldg., Pittsburgh, Pa., ALR
Wood, Edward Christopher, 405 Park Ave., and 80 Maple Ave., White Plains, N. Y., Op.
Young, Clarence M., 305 South H St., Aberdeen, Wash., ALR

Mr. President, I nominate these 141 candidates for membership.

DR. RALPH FENTON: I move that the Secretary be instructed to cast the unanimous ballot for the election of these members.

(The motion was seconded and carried, and the 141 candidates were elected members of the Academy.)

Election of Officers

DR. W. P. WHERRY: By authority of the Council, I offer the following nominations:

For President — DR. FRANK E. BURCH (ophthalmologist)
St. Paul, Minn.

(Upon motion regularly made and seconded, the nominations were closed for President, and Secretary Wherry cast the unanimous ballot for the election of Dr. Burch.)

DR. WHERRY:

For President-Elect — DR. LEE W. DEAN (otolaryngologist)
St. Louis, Mo.

(Upon motion regularly made and seconded, the nominations were closed for President-Elect, and Secretary Wherry cast the unanimous ballot for the election of Dr. Dean.)

DR. WHERRY: *For First Vice President:*

DR. GORDON F. HARKNESS (otolaryngologist)
Davenport, Iowa

(Upon motion regularly made and seconded, the nominations were closed for First Vice President, and Secretary Wherry cast the unanimous ballot for the election of Dr. Harkness.)

DR. WHERRY: *For Second Vice President:*

DR. JOHN H. DUNNINGTON (ophthalmologist)
New York, N. Y.

(Upon motion, regularly made and seconded, the nominations were closed for Second Vice President and Secretary Wherry cast the unanimous ballot for the election of Dr. Dunnington.)

DR. WHERRY: *For Third Vice President:*

DR. CARROLL SMITH (otolaryngologist)
Spokane, Wash.

(Upon motion regularly made and seconded, the nominations were closed for Third Vice President and the Secretary cast the unanimous ballot for the election of Dr. Smith.)

DR. WHERRY: The Council has deemed it unwise to change the personnel of the Comptroller or the Secretarial Group. The same will hold over, if re-elected.

PRESIDENT EAGLETON: I suggest that the President-Elect read the names of the Secretarial Group.

PRESIDENT-ELECT BURCH:

Comptroller:
DR. SECORD H. LARGE.....Cleveland, Ohio

Executive Secretary-Treasurer:
DR. WILLIAM P. WHERRY.....Omaha, Nebr.

Secretary for Ophthalmology:
DR. WILLIAM L. BENEDICT.....Rochester, Minn.

Secretary for Otolaryngology:
DR. JOHN L. MYERS.....Kansas City, Mo.

Secretary for Instruction — Ophthalmology:
DR. HARRY S. GRADLE.....Chicago, Ill.

Secretary for Miscellaneous Activities and
Acting Secretary for Instruction — Otolaryngology:
DR. RALPH A. FENTON.....Portland, Ore.

(Upon motion regularly made and seconded, the entire secretarial group was unanimously elected.)

DR. WHERRY: *For Member of the Council:*

DR. GRADY E. CLAY (ophthalmologist)
Atlanta, Ga.

(Upon motion regularly made and seconded, Dr. Clay was unanimously elected.)

DR. WHERRY: The Council has nominated and appointed as a member of the Board of Ophthalmology, DR. WALTER B. LANCASTER of Boston; and as a member of the Committee on Optics and Visual Physiology, DR. JOHN N. EVANS of Brooklyn, N. Y.

Selection of 1936 Convention City

DR. WHERRY: The Council has canvassed the picture carefully and selected New York City as the meeting place for 1936.

Introduction of President-Elect

PRESIDENT EAGLETON: Now it is my pleasure to introduce to you your new President, but before turning the meeting over to him, I want to express to each and every one of you, but especially to the secretarial group, my thanks for the uniform kindness they have shown me in an effort to make me do my job with the greatest of ease, so that this year that has passed and the associations I have made mark a milestone in my life.

I want to introduce to you a man who is much more worthy of the honor that is conferred upon him than I am, Dr. Burch.

PRESIDENT-ELECT BURCH: Members of the Academy, I have no inferiority complex — at least, I have never been accused of it — but I know my own unworthiness in assuming this office. All I can say is that I thank you and promise you I will endeavor, so far as my limited ability permits, to carry on the traditions of the Academy with my very best efforts. I thank you.

I should add that the incoming President does not take office until January 1, and Dr. Eagleton will continue in the conduct of the business affairs.

Adjournment

PRESIDENT EAGLETON: As your President, I declare this meeting adjourned.

(The meeting adjourned at six-forty o'clock.)

Directory

The American Academy
of Ophthalmology and Otolaryngology

Fortieth Annual Meeting
Cincinnati, 1935

Alphabetical List of Members

* Attended 1935 meeting. Number in parentheses indicates year in which the Fellow became a member of the Academy. Fellows are requested to supply missing data and report errors in addresses or specialties to the Executive Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska.

NOTICE: By action of the Council at the 1934 meeting:

- Fellows who have paid dues for thirty (30) years continuously will automatically become Life Members, with dues remitted.
- Fellows who have paid dues continuously for twenty-five (25) years will be termed Senior Members and their dues will be one-half the usual amount; at the present time, five dollars ($5.00) anually.
- All other Fellows (except Honorary) will be classified as Junior Members.

HONORARY FELLOWS

*Beck, Joseph C. ('01) . . . . . 185 N. Wabash Ave., Chicago, Ill., ALR
Birkett, H. S. ('06) . . . . 1190 Mountain St., Montreal, Canada, ALR
*Brunner, Hans ('35) . Gumpendorfer St. 25, Vienna, VI, Austria, ALR
Dundas-Grant, Sir James . . . . . 148 Harley St., W. 1, London, Eng.
Elliot, Lt.-Col. R. H. ('13)
54 Welbeck St., Cavendish Sq., W., London, Eng.
Elschnig, Prof. A. ('12) . Ferdinandstrasse 10, Prague, Czechoslovakia
*Foster, Hal ('94) . . . . . Professional Bldg., Kansas City, Mo., ALR
Graves, Basil ('26)
11 Devonshire Place, Upper Wimpole St., W. 1, London, Eng.
Gray, Albert A. ('11) . . . . . Glasgow, Scotland
de Grósz, Prof. Emil ('30) . . . . . Budapest, VIII, Maria, V. 39, Op.
Jackson, Edward ('02) . . . . . Republic Bldg., Denver, Colo., Op.
Johnson, Lindsay ('05)
"Fairlawn" Esplanade, Durban, Natal, Africa, Op.
Kirkpatrick, Lt.-Col. Henry ('24)
54 Welbeck St., W. 1, London, Eng.
Koller, Carl ('34) . . . . . 30 E. 58th St., New York, N. Y., Op.
Lauber, Prof. Dr. Hans ('34) . . . . Aleja Roz 8, Warsaw, Poland, Op.
Lawford, J. B. ('08) . . . Sagamore, New Road, Ashtead, Surrey, Eng.
Lister, Sir William T. ('23) . 24 Devonshire Pl., W. 1, London, Eng.
Nagar, Prof. F. ('25) . . . . . Freiestrasse 20, Zurich, Switzerland
Patterson, Maj. Gen. Robert U. ('33)
U. S. Army, Office of Surgeon General, Washington, D. C., ALR
Tilley, Herbert ('29) . . University College Hosp., London, Eng., Op.
Van der Hoeve, Prof. J. ('21) . Rijnsburgerweg, 6 A, Leiden, Holland

LIFE MEMBERS

A

*Adams, A. L.('06) . . . . .Professional Bldg., Jacksonville, Ill., OALR
Adams, C. F.('06) . . . . . .34 W. State St., Trenton, N. J., OALR
Alkire, H. L.('04) . . . . . .614 Kansas Ave., Topeka, Kan., OALR
Allen, J. H. . . . . .248 S. Michigan Ave., Pasadena, Calif., ALR
Amberg, Emil('06) . . . . . .Maccabee Bldg., Detroit, Mich., ALR
Andrews, A. H.('98) . . . .30 N. Michigan Ave., Chicago, Ill., OALR

B

Baker, Chas. H.('06) .Bay City Bank Bldg., Bay City, Mich., OALR
Ball, Michael V.('04) . . . . . .316 Hazel St., Warren, Pa., OALR
Bane, William C.('04) . . . . . .Republic Bldg., Denver, Colo., Op.
*Barnhill, John F.('03)
5369 Pine Tree Drive, Miami Beach, Fla., ALR
*Beattie, Robert('06) . .David Whitney Bldg., Detroit, Mich., OALR
Bellows, George E. ('96)
Professional Building, Kansas City, Mo., Op.
Bennett, Arthur G.('05) . .150 N. Pearl St., Buffalo, N. Y., Op.
Bernstein, E. J.(1900) . . . . . .10 Peterboro, Detroit, Mich., OALR
Blaauw, E. E.('05) . . . . . .190 Ashland Ave., Buffalo, N. Y., Op.
*Black, Melville('04) .424 Metropolitan Bldg.,Denver,Colo., Op.
*Black, Nelson Miles('03) . .Huntington Bldg., Miami, Fla., Op.
Black, W. D.(1900) . . . . .Metropolitan Bldg., St. Louis, Mo., ALR
Brobst, C. H.('06) . . . .Central Natl. Bank Bldg., Peoria, Ill., OALR
Brown, Edward J. ('11)
3027 Pleasant Ave., Minneapolis, Minn., (Retired) OALR
*Brown, John E.('01) . . . . .370 East Town St., Columbus, O., OALR
Bruner, Wm. E.('05) . . . . . .Guardian Bldg., Cleveland, O., Op.
Buckman, E. U.('06) .70 S. Franklin St., Wilkes-Barre, Pa., OALR
Burke, T. A.('06) . . . . . .Rose Bldg., Cleveland, O., OALR

C

*Carmody, T. E.('04) . . . . .Metropolitan Bldg., Denver, Colo., ALR
Carter, W. W. . . . . .140 E. 54th St., New York, N. Y., ALR
Chenery, W. E. . . .377 Commonwealth Ave., Boston, Mass., ALR
Clevenger,W. F.('05) .Hume-Mansur Bldg.,Indianapolis,Ind., ALR
Culbertson, L. R.('98) . . . .Masonic Temple, Zanesville, O., OALR

D

Dabney, S. G. ('07) . . . . . .Starks Bldg., Louisville, Ky., OALR
Davis, J. Leslie('06) . . . . . .135 S. 18th St., Philadelphia, Pa., ALR
Day, Ewing W.('08) . .121 University Place, Pittsburgh, Pa., OALR
Dean, F. W.('03) . . . . . .532 First Ave., Council Bluffs, Ia., OALR
Dennis, F. L.('07) .Ferguson Bldg., Colorado Springs, Colo., ALR
Dixon, W. E. . . . . .1413 Spurgeon, Santa Ana, Calif., OALR
Doane. L. L. ('05) . . . . . .357 N. Main St., Butler, Pa., OALR
Dodd, Oscar ('03) . . . . . .636 Church St., Evanston, Ill., Op.
Donovan, John A.('02) . . .507 Phoenix Bldg., Butte, Mont., OALR

E

*Easton, J. C.('04) . . First Nat'l Bank Bldg., Springfield, O., OALR
*Ellett, E. C.('98) . . . . . Exchange Bldg., Memphis, Tenn., Op.
Emerson, Linn('06) . . . . . Metropolitan Bldg., Orange, N. J., Op.
Engle, H. P.('02) . . . . . 212 West First Ave., Newton, Ia., OALR

F

Farrington, P. M.('04) . 899 Madison Ave., Memphis, Tenn., ALR
Ferguson, E. S.('06) . Med. Arts Bldg., Oklahoma City, Okla., OALR
Fisher, W. A.('96) . . . . 231 W. Washington St., Chicago, Ill., OALR
Flagg, J. D.('05) . . . . . 158 Anderson Place, Buffalo, N. Y., Op.A
*Frank, Ira('05) . . . . . 104 S. Michigan Blvd., Chicago, Ill., ALR
Friedenwald, Harry('04) . . 1212 Eutaw Pl., Baltimore, Md., Op.A
Fringer, W. R.('04) . . . William Brown Bldg., Rockford, Ill., Op.

G

Gallaher, T. J.('04) . . . . . California Bldg., Denver, Colo., ALR
Geiger, C. W. ('04) . . . . . Arcade Bldg., Kankakee, Ill., OALR
*Goldstein, M. A.('96) . . . . 4575 W. Papin St., St. Louis, Mo., ALR
Green, John('05) . . 3720 Washington Blvd., St. Louis, Mo., Op.
Grushlaw, Israel('05)
271 Central Park West, New York, N. Y., ALR

H

*Harris, W. C.(1900) . . . . . Groton Bldg., Cincinnati, O., ALR
Harvey, N. D.('04) . . . . 112 Waterman St., Providence, R. I., OALR
Hawley, C. W. . . . . Medical Arts Bldg., Chicago, Ill., OALR
Heckel, Edward B.('02) . . . 500 Penn Ave., Pittsburgh, Pa., Op.
*Heitger, J. D.('05) . . . . . Heyburn Bldg., Louisville, Ky., OALR
Hess, Wm. L. ('04)
1601 Sawtelle Blvd., West Los Angeles, Calif., OALR
Hetzel, C. C.('06) . . . . . Eccles Bldg., Ogden, Utah, OALR
Hitz, H. B.(1900) . . . . . Bankers Bldg., Milwaukee, Wis., ALR
Holt, E. E., Jr.('04) . . . . . 723 Congress St., Portland, Me., Op.

I

Ingersoll, J. M.('08) . . 1700 S. Bayshore Lane, Miami, Fla., ALR

J

*Jackson, Chevalier('04) . 3432 N. Broad St., Philadelphia, Pa., L
Jean, George W. . . . . 7 E. Valerio, Santa Barbara, Calif., Op.

K

*Kincaid, J. H.('03) . . . . . 421 Church Ave., Knoxville, Tenn., OALR
King, G. L.('06) . . . . . 537 E. Market St., Alliance, O., OALR
*Kirkpatrick, Samuel('03) . . 902 Alabama Ave., Selma, Ala., OALR
Klinedinst, John F.('06) . . . . 222 S. George St., York, Pa., Op.A

L

LAMB, R. S.('06) . . . . . Stoneleigh Court, Washington, D. C., Op.
LAPSLEY, ROBERT M.('02) . . . . Masonic Temple, Keokuk, Ia., OALR
*LARGE, S. H.('04) . . . . . Hanna Bldg., Cleveland, O., ALR
LEVY, ROBERT('96) . . . . . Metropolitan Bldg., Denver, Colo., ALR
*LEWIS, F. PARK('04) . . . . . 454 Franklin St., Buffalo, N. Y., Op.
LOCHARD, L. B. . . . . Metropolitan Bldg., Denver, Colo., OALR
LUKENS, CHARLES('06) . . . . . 316 Michigan St., Toledo, O., OALR

M

McHENRY, D. D.('12) . Med. Arts Bldg., Oklahoma City, Okla., OALR
*McREYNOLDS, JOHN O.('03)
907 Mercantile Bank Bldg., Dallas, Texas, OALR
MATHENY, R. C.('04) . . . . . 306 E. Main St., Galesburg, Ill., OALR
MAY, CHAS. H.('04) . . . 698 Madison Ave., New York, N. Y., Op.
*MEIERHOF, EDWARD L.
care Guaranty Trust Co., New York, N. Y., OALR
MERCER, RICHARD E.('03) . . D. Whitney Bldg., Detroit, Mich., LR
*MILLETTE, J. W.('06) . . . . . 117 S. Main St., Dayton, O., OALR
MINER, S. G.('96) . . . . 2641 E. Jefferson Ave., Detroit, Mich., ALR
*MINOR, C. L.('02) . . . . First Nat'l Bank Bldg., Springfield, O., OALR
MONOSMITH, O. B.('05) . . . . . O. P. S. Bldg., Lorain, O., OALR
*MOORE, T. W.('01) . . . . 1050 Fifth Ave., Huntington, W. Va., OALR
MOULTON, H. (1900)
Merchants National Bank Bldg., Ft. Smith, Ark., OALR

N

NANCE, WILLIS O. . . . . 5242 Hyde Park Blvd., Chicago, Ill., OALR
*NEWHART, HORACE('05) . Med. Arts Bldg., Minneapolis, Minn., ALR
NOONAN, W. J.('05) . . . . . 4753 Broadway, Chicago, Ill., OALR

O

OWEN, FRANK S. . . . 44 El Bosque Rd., Santa Barbara, Calif., OALR

P

PFAFFLIN, C. A.('03) . Bankers Trust Bldg., Indianapolis, Ind., OALR
PFINGST, A. O.('99) . . . . . Heyburn Bldg., Louisville, Ky., Op.
*PHINNEY, F. D.('05) . . . . . 22 W. 7th St., Cincinnati, O., OALR

R

RINDLAUB, JOHN H.('06) . . de Lendrecie Bldg., Fargo, N. D., OALR
ROBERTS, WM. H.('03) . 65 N. Madison Ave., Pasadena, Calif., Op.
ROLLER, LOUIS A. ('03)
446 Paris Ave., S.E., Grand Rapids, Mich., Retired

S

SANDERSON, H. H.('06) . David Whitney Bldg., Detroit, Mich., OALR
SAUER, W. E.('04) . . . . Beaumont Med. Bldg., St. Louis, Mo., ALR
SCALES, J. W.('96) . . . . . 204½ Main St., Pine Bluff, Ark., OALR

DE SCHWEINITZ, GEORGE E.('04)
1705 Walnut St., Philadelphia, Pa., Op.
SELFRIDGE, G. L. .....384 Post St., San Francisco, Calif., ALR
SHOEMAKER, W. A.('96) .1006 Carleton Bldg., St. Louis, Mo., Op.
SHREVE, OWEN M.('05) .....162 W. 8th St., Erie, Pa., Op.
*SHURLY, BURT R.('03) ..62 Adams Ave., W., Detroit, Mich., ALR
*SLEIGHT, R. D.('06) .Security Nat'l Tower, Battle Creek, Mich., Op.
SNYDER, W. H.('06) .....2001 Collingwood Ave., Toledo, O., OALR
SPALDING, J. A.('04) .....627 Congress St., Portland, Me., Op.A
*SPENCER, FRANK R.('04) ...Physicians Bldg., Boulder, Colo., OALR
STEIN, O. J.(1900) ..... Palos Verdes Estates, Calif., ALR
*STEWART, T. M. .....901 Union Trust Bldg., Cincinnati, O., OALR
STIEREN, EDWARD('04) .626 Union Trust Bldg., Pittsburgh, Pa., Op.
STILLSON, H. .....Seabord Bldg., Seattle, Wash., OALR
STRADER, GEO. L.('04) ...408 Hynds Bldg., Cheyenne, Wyo., OALR
STROUT, E. S.('06) ..Medical Arts Bldg., Minneapolis, Minn., OALR
STUEBER, F. G.('05) .....209 W. North St., Lima, Ohio, OALR

T

TAYLOR, LILLIAN E.('05)
R.F.D. No. 1, Box 175, Carmel-by-the-Sea, Calif., ALR
THOMSEN, G. VON COLDITZ('06) .25 E. Washington, Chicago, Ill., OALR
TIMBERMAN, ANDREW('03) .....Beggs Bldg., Columbus, O., OALR
TOMS, S. W. S.('09) .....120 S. Broadway, Nyack, N. Y., OALR

V

VEASEY, C. A., JR.('06)
Paulson Medical and Dental Bldg., Seattle, Wash., OALR
VINSONHALER, F.('02) ....Urquhart Bldg., Little Rock, Ark., OALR

W

WEEKS, JOHN E. ('04)
360 Edgecliff Road, Portland, Ore. (Retired) Op.
WELLS, DAVID W.('08) ...Hotel Westminster, Boston, Mass., Op.
WILBUR, EDWARD P.('03)
American Nat'l Bank Bldg., Kalamazoo, Mich., OALR
WOOD, CASEY A.('96) .....7 W. Madison St., Chicago, Ill., Op.
WOODRUFF, T. A.('96) ...309 State St., New London, Conn., Op.
WURDEMANN, HARRY V. ('99)
320 Stimson Bldg., Seattle, Wash., Op

SENIOR MEMBERS

A

ALDERDYCE, WM. W.('08) . . . .513 Madison Ave., Toledo, O., OALR

B

BAILEY, HAROLD ('08) . . . . . Landers Bldg., Springfield, Mo., OALR
BALLIN, M. J.('08) . . . . . 20 E. 76th St., New York, N. Y., ALR
BARDENHEIER, F. G. A.('09) . . . Frisco Bldg., St. Louis, Mo., ALR
BARKER, O. G. A. ('09)
804 Johnstown Bank and Trust Co., Johnstown, Pa., Op.
BEEBE, C. S.('07) . . 324 E. Wisconsin Ave., Milwaukee, Wis., Op.
*BERRY, D. F.('11) . . . . Bankers Trust Bldg., Indianapolis, Ind., ALR
*BLEDSOE, R. W.('07) . . . . 1005 Madison Ave., Covington, Ky., OALR
*BRAWLEY, F. E.('07) . . . . 30 N. Michigan Blvd., Chicago, Ill., Op.
BROWN, J. M.('11) . . . . . 1136 W. 6th St., Los Angeles, Calif., ALR
BROWN, SAMUEL H.('10) . 1930 Chestnut St., Philadelphia, Pa., Op.
BRUDER, JOSEPH('08) . . . . . 57 W. 57th St., New York, N. Y., OALR
BRYANT, ALICE G.('08) . . 405 Marlborough St., Boston, Mass., ALR
*BURCH, FRANK E.('07) . . . . . Hamm Bldg., St. Paul, Minn., Op.
BUTT, MIRIAM M.('08) . . 2045 Chestnut St., Philadelphia, Pa., Op.

C

CALHOUN, F. PHINIZY('08) . 131 Doctors' Bldg., Atlanta, Ga., Op.
CAMPBELL, DON M.('08) . David Whitney Bldg., Detroit, Mich., OALR
CARR, GEO. W.('09) . . . 54 S. Franklin St., Wilkes-Barre, Pa., OALR
*COHEN, MARTIN('10) . . . . . 29 E. 64th St., New York, N. Y., Op.
COHN, FELIX('11) . . . . 945 West End Ave., New York, N. Y., ALR
COLLINS, E. W.('10) . . . . . 1578 Humboldt St., Denver, Colo., ALR
CREIGHTON, WM. J.('10) . Physicians Bldg., Philadelphia, Pa., Op.
*CULLOM, M. M.('10)
1119 Bennie-Dillon Bldg., Nashville, Tenn., OALR

D

DAVIS, A. EDWARD('08) . . . . 40 E. 61st St., New York, N. Y., Op.
DENNIS, GEO. J.('10) . . . . 25 E. Washington St., Chicago, Ill., ALR

G

GARBER, J. M.('11) . . . . . 14 N. Mulberry St., Mansfield, O., OALR
GODDARD, H. M.('11) . . . . . 1531 Spruce St., Philadelphia, Pa., ALR
GOLDBACK, L. J.('11) . . . . 1012 N. Charles St., Baltimore, Md., Op.
*GRADLE, HARRY S.('11) . . . 58 E. Washington St., Chicago, Ill., Op.
*GREENWOOD, ALLEN('11)
82 Commonwealth Ave., Boston, Mass., Op.
GUTHRIE, A. L. ('10)
1200 N. Walker St., Oklahoma City, Okla., OALR

H

*HALL, G. C.('07) . . . . . 705 Brown Bldg., Louisville, Ky., OALR
*HARKNESS, GORDON F.('11) . . . . Putnam Bldg., Davenport, Ia., OALR

HARRIS, C. M. ('10)
604 Johnstown Trust Bldg., Johnstown, Pa., OALR
*HARRIS, H. B.('10) . . . . . Fidelity Medical Bldg., Dayton, O., OALR
HATCH, C. B.('08) . . . . . 3 W. Church St., Newark, O., OALR
*HAUGHEY, WILFORD H.('11) . . Post Bldg., Battle Creek, Mich., OALR
*HAYDEN, AUSTIN A.('10) . 25 E. Washington St., Chicago, Ill., OALR
HEARD, MARY K.('10)
128—19th Ave., St. Petersburg, Fla. (Retired), Op.
HENNING, CARL('08) . 1835 Eye St., N.W., Washington, D. C., Op.
*HOGUE, D. W.('08) . . . . . 34 W. High St., Springfield, O., OALR
HOPKINS, W. E.('07) . 321 S. Lorraine Blvd., Los Angeles, Calif., OALR
HURD, L. M.('10) . . . . . 39 E. 50th St., New York, N. Y., ALR

I

INGERMAN, S. M.('09) . 418 Central Pk.W., New York, N. Y., OALR
IRWIN, V. J., SR.('09) . . . . 1634 Main St., Springfield, Mass., OALR
*IVINS, H. M.('10)
815 Merchants Nat'l Bank Bldg., Cedar Rapids, Ia., OALR

J

JARECKY, HERMAN('08) . . . 120 W. 86th St., New York, N. Y., OALR
JOBSON, G. B.('08) . . West Park & Liberty Sts., Franklin, Pa., OALR

K

*KING, CLARENCE('10) . . . Union Central Bldg., Cincinnati, O., Op.
KNAPP, ARNOLD('08) . . . . . 10 E. 54th St., New York, N. Y., Op.
*KOPETZKY, S. J.('08) . . . . . 51 W. 73rd St., New York, N. Y., ALR
KRIEGER, GEO. M.('10) . 701 Washington St., Michigan City, Ind., OALR
KRUG, ERNEST F.('08) . . . . 988 Fifth Ave., New York, N. Y., Op.

L

LAFORCE, E. F.('08) . Iowa State Bank Bldg., Burlington, Ia., OALR
LESTER, HARRY S.('09) . . . . . 420 Murray Bldg., Streator, Ill., OALR

M

MCALLISTER, J. C.('08) . . . . . 10 S. Mill Ave., Ridgway, Pa., AL
*MCCASKEY, CARL H.('11) . Guaranty Bldg., Indianapolis, Ind., ALR
MCCOOL, J. L.('11) . . . . . 450 Sutter St., San Francisco, Calif., Op.
*MCKEE, S. HANFORD('08) . 1528 Crescent St., Montreal, Can., Op.
*METZ, ROY B.('09) . . . . . Hanna Bldg., Cleveland, O., Op.
*METZENBAUM, MYRON ('08)
Carnegie Medical Bldg., Cleveland, O., Op.
*MOLT, WM. F.('11) . . Bankers Trust Bldg., Indianapolis, Ind., ALR
MONSON, S. H.('10) . . . 1322 B. F. Keith Bldg., Cleveland, O., OALR

O

*OBERG, C. M.('10) . . . . . Syndicate Bldg., Minneapolis, Minn., OALR
*OVERMAN, F. V.('11) . Hume-Mansur Bldg., Indianapolis, Ind., ALR

P

PAYNE, A. L.('08) . . . . . 119 S. Barston St., Eau Claire, Wis., OALR
*PEARSON, WM. W. ('08)
417 Bankers Trust Bldg., Des Moines, Ia., OALR
*POLLOCK, H. L.('11) . . . . . 2561 N. Clark St., Chicago, Ill., ALR
PRATT, JOHN A.('10)
Physicians and Surgeons Bldg., Minneapolis, Minn., OALR
PRICE, NORMAN W.('08) . 445 Third St., Niagara Falls, N. Y., OALR

R

RAVDIN, M.('11) . . Central Union Bank Bldg., Evansville, Ind., OALR
*REGER, H. S.('08) . . . . . Physicians Bldg., Jamestown, N. Y., OALR
REYNOLDS, H. G.('09) . . Citizens Sav. Bk. Bldg., Paducah, Ky., OALR
RINDLAUB, ELIZABETH P. ('10)
de Lendrecie Bldg., Fargo, N. D., OALR
RINGLE, CHAS. A.('07) . . . . . 912 Ninth Ave., Greeley, Colo., ALR
*ROW, GEORGE S.('11) . Hume-Mansur Bldg., Indianapolis, Ind., Op.
RUBY, F. McK.('07) . . . . . 235 W. Pearl St., Union City, Ind., OALR
RUST, E. G.('07)
Wade Park Manor, E. 107th and Lane Park, Cleveland, O., Op.

S

*SAUER, W. W.('10) . . . . . 231 Third St., Marietta, O., OALR
*SEWALL, E. C.('10) . .
Stanford Univ. Hospital, San Francisco, Calif., ALR
SHASTID, T. H.('09) . . . . 202 W. Superior St., Duluth, Minn., Op.
SIMPSON, W. LIKELY('11)
Physicians and Surgeons Bldg., Memphis, Tenn., ALR
SMITH, DORLAND('10) . . 881 Lafayette St., Bridgeport, Conn., Op.A
*STANBERY, HENRY('10) . . . The Doctors Bldg., Cincinnati, O., Op.
STILES, CHAS. M.('08) . . 1831 Chestnut St., Philadelphia, Pa., Op.

T

*THOMPSON, E. H.('07) . . . . . 19 W. 7th St., Cincinnati, O., OALR
THOMPSON, G. H.('09) . . . . . 74 North St., Pittsfield, Mass., OALR
THOMSON, J. J.('07) . 531 E. Lincoln Ave., Mt. Vernon, N. Y., ALR
*TIVNEN, RICHARD J.('08) . 30 N. Michigan Ave., Chicago, Ill., OALR
TUCKERMAN, W. H.('08) . . . 733 Osborn Bldg., Cleveland, O., OALR
*TURNER, OLIVER W.('09) . . . . Masonic Temple, Augusta, Me., OALR

U

UPHAM, HELEN F.('07) . . . 305 3rd Ave., Asbury Park, N. J., OALR

W

*WEED, HARRY M.('08) . . . . 196 Linwood Ave., Buffalo, N. Y., Op.
*WHERRY, W. P.('10) . . . 1500 Med. Arts Bldg., Omaha, Neb., OALR
WIENER, ALFRED('08) . . . . . 550 Park Ave., New York, N. Y., OALR
*WILLIAMS, W. C.('08) . . . . . Jefferson Bldg., Peoria, Ill., OALR
*WYLIE, C. B.('10) . . . . . 269 High St., Morgantown, W. Va., OALR

JUNIOR MEMBERS

A

ABBOTT, W. J.('19) . . . Carnegie Medical Bldg., Cleveland, O., OALR
*ABRAHAMSON, IRA A.('27) . Union Trust Bldg., Cincinnati, O., Op.
*ADAMS, CHARLES JOSEPH('28)
Armstrong-Landon Bldg., Kokomo, Ind., OALR
ADAMS, D. S. . . . . Hume-Mansur Bldg., Indianapolis, Ind., OALR
ADAMS, ELDRIDGE S.('20)
Central Aviation School, Hangchow, China, ALR
ADAMS, FRANK M.('24) . 122 Waterman St., Providence, R. I., ALR
ADAMS, FREDERICK W. ('20)
Roosevelt Clinic, 1114 Boylston Ave., Seattle, Wash., OALR
*ADKINS, GEO. ELI('29) . . . . Jackson Infirmary, Jackson, Miss., ALR
*ADLER, FRANCIS HEED('29) . 313 S. 17th St., Philadelphia, Pa., Op.
AEBLI, RUDOLF('31) . . . . . 30 East 40th St., New York, N. Y., Op.
AGAN, WM. B. ('30) . . . . . 510 Third St., Brooklyn, N. Y., Op.
AINSLIE, GEORGE ('21) . . . . . Selling Bldg., Portland Ore., OALR
ALBRIGHT, GEO. C.('31) . Johnson Co. Bk. Bldg., Iowa City, Ia., ALR
ALBRIGHT, P. M.('22), Lt.-Com., M. C., U. S. Navy,
Naval Hospital, Brooklyn, N. Y., OALR
ALDEN, ARTHUR M.('22) . . . . . Frisco Bldg., St. Louis, Mo., ALR
ALEXANDER, GEO. J.('23) . 1930 Chestnut St., Philadelphia, Pa., ALR
ALEXANDER, LUCIEN W.('34)
1230 Maison Blanche Bldg., New Orleans, La., ALR
ALEXANDER, SAMUEL A.('33)
1801 Eye St., N.W., Washington, D. C., ALR
ALEXANDER, S. L.('33) . Med Arts Bldg., Toronto, Ont., Can., ALR
*ALFARO, V. R.('31) . . 1801 Eye St., N.W., Washington, D. C., ALR
ALLAN, WM. B.('34) . . . . . 410 Park Ave., New York, N. Y., Op.
ALLEN, CHARLES E.('20)
712 Waldheim Bldg., Kansas City, Mo., OALR
ALLEN, G. HENRY('16) . . . . . Mills Bldg., Topeka, Kan., OALR
ALLEN, G. V. ('21) . . . . . 835 Kansas Ave., Topeka, Kan., OALR
*ALLEN, ORRIS T. ('28)
422 Rose Dispensary Bldg., Terre Haute, Ind., OALR
*ALLEN, THOMAS DYER('28) . 122 S. Michigan Ave., Chicago, Ill., Op.
*ALMOUR, RALPH('25) . . . . 51 W. 73rd St., New York, N. Y., ALR
ALTRINGER, A. N.('20) . Professional Bldg., Kansas City, Mo., ALR
*ALVIS, BENNETT Y.('28) . . 308 N. 6th St., St. St. Louis, Mo., Op.
AMANO, K. W. ('29) . . . . . 132 W. 1st St., Los Angeles, Calif., ALR
AMBROSE, ELMER C.('27) . . . . . 910 Main St., Trenton, Mo., OALR
ANDERSON, CARL M. ('26) . . . . Mayo Clinic, Rochester, Minn., ALR
ANDERSON, W. B.('13) . 309 W. Anderson St., Brownwood, Tex., OALR
ANDERSON, WM. B.('35) . . McPherson Hosp., Durham, N. C., Op.
ANDREW, JAMES H.('32) . . 163 Hancock St., Brooklyn, N. Y., Op.
ANDREWS, B. F.('12) . . . . 25 E. Washington St., Chicago, Ill., OALR
ANDREWS, H. D. ('12) . . . . . 33 Allen St., Buffalo, N. Y., Op.
ANDREWS, M. P.('15) . . . . . 826 S. 8th St., Manitowoc, Wis., OALR

ANNON, W. T.('23).....235 S. 15th St., Philadelphia, Pa., ALR
ANTHONY, DANIEL HARBERT ('29)
Exchange Bldg., Memphis, Tenn., Op.
APPLEMAN, L. F. ('13)...308 S. 16th St., Philadelphia, Pa., Op.
ARBUCKLE, DAVID S.('31).....625 W. Main St., Kent, Ohio, OALR
*ARBUCKLE, MILLARD F....539 N. Grand Ave., St. Louis, Mo., ALR
ARMSTRONG, E. LESLIE('32)..205 W. 2nd St., Duluth, Minn., ALR
*ARMSTRONG, R. M.('28)..1st Nat'l Bk. Bldg., Lexington, Ky., OALR
ARNOLD, FRANCIS J.('12).....182 Pearl St., Burlington, Vt., OALR
*ARONSON, SIDNEY S.('29)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
ARRELL, W.('21).....317 Main St. E., Hamilton, Ontario, OALR
ASBILL, DAVID ST. PIERRE('33)
1512 Marion St., Columbia, S. C., Op.
*ASBURY, MARY K.('25)..2466 Grandin Road, Cincinnati, O., Op.
ASHLEY, T. W.('20).....5711 Eighth Ave., Kenosha, Wis., OALR
ATHENS, A. G.('32).....Medical Arts Bldg., Duluth, Minn., Op.
ATKINSON, D. T. ('18)
827 Med. Arts Bldg., San Antonio, Tex., OALR
ATKINSON, THOMAS E. ('29).Coronado Bldg., Greeley, Colo., ALR
*ATKINSON, WALTER S.('25)
168 Sterling St., Watertown, N. Y., Op.
ATWOOD, ED A. ('32).....360 Park Ave., Paterson, N. J., ALR
AUERBACH, JULIUS.....1016 Fifth Ave., New York, N. Y., ALR
AVERY, ELROY J.('34)..11 N. Goodman St., Rochester, N. Y., ALR
AYNESWORTH, H. T.('12)...Medical Arts Bldg., Waco, Tex., OALR

B

BABBITT, JAMES A.('24)..1912 Spruce St., Philadelphia, Pa., ALR
BAER, BENJAMIN F., JR. ('31)
1927 Spruce St., Philadelphia, Pa., Op.
*BAESEMAN, R. WINFIELD('29)
501 Grand Ave., Asbury Park, N. J., ALR
*BAHN, CHAS. A.('22).Pere Marquette Bldg., New Orleans, La., Op.
BAILEY, FRED W.('12)
Merchants Nat'l Bank Bldg., Cedar Rapids, Ia., OALR
BAIRD, CHAS. G.('20)....Theatre Bldg., Santa Maria, Calif., OALR
BAKER, HARRY B.('16).....59 Main St., Taunton, Mass., OALR
*BALDWIN, H. E. ('30).....Temple Bldg., Danville, Ill., ALR
BALENTINE, P. L.('15)..1524 Chestnut St., Philadelphia, Pa., Op.
BALLENGER, H. C.('15)...25 E. Washington St., Chicago, Ill., ALR
BALLON, D. H.('24)....1471 Crescent St., Montreal, Canada, ALR
BANE, W. M. .....Republic Bldg., Denver, Colo., Op.
BARBER, F.('13).....75 S. Fitzhugh St., Rochester, N. Y., Op.
*BARKAN, HANS .....2351 Clay St., San Francisco, Calif., Op.
BARKAN, OTTO('29).....490 Post St., San Francisco, Calif., Op.
BARKER, CHAS. B.('20).224½ W. Okla. Ave., Guthrie, Okla., OALR
BARKHORN, CHAS.W.('33).223 Roseville Ave., Newark, N. J., ALR
BARLOW, ROY A.
Medical Arts Bldg., 277 Alexander St., Rochester, N. Y., ALR

BARNES, LANCELOT('35) .170 St. George St., Toronto, Canada, Op.
*BARNETT, IRVING F.('33) . . .1005 Belmont Ave., Chicago, Ill., ALR
BARNETT, SAUL E. ('29) . . . . . .25 Parson St., Detroit, Mich., ALR
BARON, S. H.('35) . . . . . .309 State St., New London, Conn., ALR
BARR, A. S. ('32) . . .1st Nat'l Bank Bldg., Ann Arbor, Mich., Op.
BARTON, JOHN F.('31) . . .1346—14th Ave., Longview, Wash., ALR
BARTON, WM. C. ('32)
104½ W. San Francisco St., Santa Fe, N. M., Op.
*BASS, A. L.('24) . . . . . .Heyburn Bldg., Louisville, Ky., OALR
BASSEN, ED. J.('30) . . . .654 Madison Ave., New York, N. Y., Op.
*BATSON, OSCAR V.('34), Graduate School of Medicine,
University of Pennsylvania, Philadelphia, Pa., ALR
BAUM, H. L. ('17) . . . . . .510 Republic Bldg., Denver, Colo., ALR
BAUM, WM. W.('30) . . . . . .Guardian Bldg., Salem, Ore., ALR
BEACH, B. S. ('27) . . . . . .140 W. 86th St., New York, N. Y., Op.
*BEACH, S. JUDD('14) . . . . . .704 Congress St., Portland, Me., Op.
BEAL, HOMER A.('26) . .Professional Bldg., Kansas City, Mo., ALR
*BEALS, C. WEARNE ('30) . . . . . .41 N. Brady St., DuBois, Pa., ALR
BEATTIE, WILLIAM H. ('21) . .258 Genesee St., Utica, N. Y., OALR
*BEATTY, H. G.('16) . . . . . .681 E. Broad St., Columbus, O., ALR
BEAUDOUX, HENRY A. . . . . .230 Grand Ave., Oakland, Calif., OALR
*BECK, A. L. ('27) . . .421 Huguenot St., New Rochelle, N. Y., OALR
BECK, FREDERICK L. ('20) .408 Hynds Bldg., Cheyenne, Wyo., ALR
BECK, ROBT. J.('35) . . . . . .105 Main St., Saranac Lake, N. Y., Op.
BEDELL, ARTHUR J. ('12) . . . . . .344 State St., Albany, N. Y., Op.
BEEM, IONE F.('16) . . . . . .31 N. State St., Chicago, Ill., OALR
BEGLE, HOWELL LLEWELLYN('28)
David Whitney Bldg., Detroit, Mich., Op.
*BEHEN, WM. C.('33) . .221 N. Capitol Ave., Lansing, Mich., ALR
BEHRENS, ALVIN ('33) .9 Prospect Pk. West, Brooklyn, N. Y., Op.
BEIDEMAN, J. E. ('29)
Montgomery Trust Arcade, Norristown, Pa., Op.
*BEIL, J. WALLACE('18) . . . . .Bryant Bldg., Kansas City, Mo., OALR
BEISBARTH, CARL C.('31)
3720 Washington Ave., St. Louis, Mo., Op.
BELL, WILL OTTO('28) .Medical-Dental Bldg., Seattle, Wash., Op.
BELLIN, JOSEPH . . . . . .523 W. 6th St., Los Angeles, Calif., OALR
BELLOWS, JOHN('35) . . . . . .58 Washington St., Chicago, Ill., Op.
BENDER, H. A.('30) . . . . . .721 Black Bldg., Waterloo, Ia., ALR
BENDER, M. S.('30) . . . . . .88-05 150th St., Jamaica, N. Y., ALR
Also 30 E. 40th St., New York, N. Y.
*BENEDICT, WILLIAM L.('19) . .Mayo Clinic, Rochester, Minn., Op.
*BENTLEY, NEIL I.('22) .David Whitney Bldg., Detroit, Mich., OALR
*BERENS, CONRAD('22) . . . . . .35 E. 70th St., New York, N. Y., Op.
BERGMANN, M. B.('32) .959 Bushwick Ave., Brooklyn, N. Y., Op.
*BERKE, RAYNOLD N.('34) . .430 Union St., Hackensack, N. J., Op.
and 30 W. 59th St., New York, N. Y.,
*BERLINER, MILTON L.('31) .57 W. 57th St., New York, N. Y., Op.
BERMAN, PHILIP G.('35) . . . . .174 Central St., Lowell, Mass., Op.
BERNATZ, C. F.('17) . . . . . .Park Bldg., Pittsburgh, Pa., Op.

BERNHEIMER, L. BENNO('30)
104 S. Michigan Ave., Chicago, Ill., ALR
BERNSTEIN, PHILIP F. ('31)
1265 President St., Brooklyn, N. Y., ALR
BERNS, CHARLES('35).....Osborn Bldg., Cleveland, O., ALR
BERRISFORD, PAUL D.('23)
Lowry Med. Arts Bldg., St. Paul, Minn., OALR
BERRY, GORDON ('30).....36 Pleasant St., Worcester, Mass., ALR
BEST, L. K.('35).....151 Rock St., Fall River, Mass., Op.
*BEYER, A. G. ('25).....Doctors' Bldg., Cincinnati, O., OALR
BEYER, LOUIS J. ('18)....449 Delaware Ave., Buffalo, N. Y., ALR
*BEYER, THEODORE E.('31)..206 Sixteenth St., Denver, Colo., ALR
BILCHICK, EDWIN B.('35)..470 Park Ave., New York, N. Y., ALR
*BILLINGS, ROBT. J.('35)....500 Penn. Ave., Pittsburgh, Pa., Op.
BINGER, H. E.('17)...Lowry Medical Bldg., St. Paul, Minn., OALR
*BINKLEY, ROY S. ('17).....Fidelity Bldg., Dayton, O., OALR
BIRSNER, LOUIS J. ('26).....Missouri Bldg., St. Louis, Mo., ALR
BISHOP, VERNON L.('33).277 Alexander St., Rochester, N. Y., ALR
BLACK, PAUL('27).....138 N. 13th St., Lincoln, Nebr., Op.
BLACK, W. BYRON('31).Professional Bldg., Kansas City, Mo., ALR
*BLACKBURN, W. J.('19).....663 Reibold Bldg., Dayton, O., OALR
*BLACKMAR, FRANCIS B('31).Swift Kyle Bldg., Columbus, Ga., ALR
BLAIR, MORTIMER WARREN('28)
N. E. cor. 20th and Chestnut Sts., Philadelphia, Pa., Op.
BLAIR, W. W.('17)....121 University Place, Pittsburgh, Pa., Op.
BLAKESLEY, T. S. ....Professional Bldg., Kansas City, Mo., OALR
BLASSINGAME, C. D.('23).899 Madison Ave., Memphis, Tenn., ALR
*BLICKENSTAFF, A. J.('22)...Alliance Life Bldg., Peoria, Ill., OALR
*BLOMBERG, THORSTEN EMIL('33).501 7th St., Rockford, Ill., ALR
BLONDER, EDWIN J.('34)..185 N. Wabash Ave., Chicago, Ill., ALR
BLOOM, C. HUGH ('32).....65 N. 3rd St., Easton, Pa., ALR
BLUE, J. B.('16).....62 N. Main St., Memphis, Tenn., OALR
BLUM, HENRY N.('14).American Bk. Bldg., New Orleans, La., Op.
BOBB, E. V.('18).....2 Garfield Ave., Alhambra, Calif., OALR
*BOCHNER, MAXWELL K.('33)
329 Bloor St. West, Toronto, Canada, Op.
BOECKMANN, EGIL..Lowry Med. Arts Bldg., St. Paul, Minn., OALR
BOEHRINGER, H. WINFIELD
700 Central Medical Bldg., Philadelphia, Pa., OALR
BOEMER, L. C.('30)...3720 Washington Blvd., St. Louis, Mo., ALR
*BOICE, RALPH E.('35).....230 Michigan St., Toledo, O., Op.
BOILER, W. F.('12).....Dey Bldg., Iowa City, Ia., OALR
BOLOTOW, NATHAN A. ('29)
126 Waterman St., Providence, R. I., ALR
BONE, HERMAN D.('20).....19 Pleasant St., Gardner, Mass., OALR
BONNER, WM. F.('26)..Medical Arts Bldg., Wilmington, Del., OALR
BOONE, J. C.('21).....Sherland Bldg., South Bend, Ind., ALR
BOONE, J. L. ('24).....Professional Bldg., Jacksonville, Fla., ALR
BORDLEY, JAMES, JR.('18).330 N. Charles St., Baltimore, Md., OALR
BORGESON, E. J.('30).Medical Arts Bldg., Minneapolis, Minn., Op.

Boss, Eugene Geo.('33)..1537 Main St., Springfield, Mass., Op.
Bostwick, D. S.('34).....106 Linwood Ave., Ardmore, Pa., ALR
Bothman, Louis('30)...122 S. Michigan Ave., Chicago, Ill., Op.
Boucher, R. B. ....Medical Dental Bldg., Vancouver, B. C., OALR
Bourbon, Oliver P.('20).520 W. 7th St., Los Angeles, Calif., OALR
Bousquet, J. P. E.('24)
66 Sherbrooke St., W., Montreal, Canada, OALR
Bouvy, Harry M.('20)
921 S. W. Washington St., Portland, Ore., OALR
*Bouvy, L. B. ('27)
Bouvy Eye and Ear Hospital, La Grande, Ore., OALR
Bowers, Chester H.('31).1136 W. 6th St., Los Angeles, Calif., ALR
Boyce, G. H.('12).....Box 417, Iron Mountain, Mich., OALR
Boyce, Wm. A.('31)...Roosevelt Bldg., Los Angeles, Calif., Op.
Boyd-Snee, Harry.....716 J. M. S. Bldg., South Bend, Ind., ALR
Boyes, T. L.('32).....654 Madison Ave., New York, N. Y., Op.
Bozer, Herrmann Eugene('23)
468 Delaware Ave., Buffalo, N. Y., ALR
Bradley, Basil V.('35).Medical Arts Bldg., Toronto, Canada, ALR
Bradley, Jeter C.('34)
900 17th St., N.W., Washington, D. C., ALR
Branigan, C. Hugh('33)..399 Park Ave., New York, N. Y., Op.
Braswell, James C.('22).1109 Med. Arts Bldg., Tulsa, Okla., OALR
Braunlin, Wm. H.('20).Marion Nat'l Bk. Bldg., Marion, Ind., OALR
Brawner, Leon Ed. ('29)
Medical Arts Bldg., 384 Peachtree, N. E., Atlanta, Ga., ALR
Bredlau, Ernest A.('29)....2753 North Ave., Chicago, Ill., ALR
Breeding, Earle G.('32)
1801 Eye St., N.W., Washington, D. C., ALR
Brehmer, Harrison L.('20)
First Nat'l Bank Bldg., Albuquerque, N. M., OALR
*Bressler, J. L.('34)....6254 S. Ashland Ave., Chicago, Ill., Op.
Breuning, Paul H.('32)..133 E. 58th St., New York, N. Y., ALR
Brewer, John D. ('20)
Baird Brewer General Hospital, Dyersburg, Tenn., OALR
Brewster, H. F.('32).Union Indem. Bldg., New Orleans, La., ALR
*Briant, Thomas E. ('29)....28 E. Main St., Welland, Ont., ALR
Bribach, Eugene J.('19)....Simpson Bldg., Atchison, Kan., OALR
Bricker, Sacks ('31)..3701 N. Broad St., Philadelphia, Pa., ALR
*Bricker, Daniel W.('21)..Uhler Phillips Bldg., Marion, O., OALR
Briggs, F. W.....Masonic Temple, Havre, Mont., OALR
*Bristow, Walter J.('35)..1512 Marion St., Columbia, S. C., ALR
Britten, G. S. ('07)....Medical Arts Bldg., Syracuse, N. Y., ALR
Brobeck, von Haller('32)
Ferguson Bldg., Colorado Springs, Colo., Op.
Brodrick, Frank W. .....301 First Ave., Sterling, Ill., OALR
*Brook, Jeffrey J.('34)
231 W. Wisconsin Ave., Milwaukee, Wis., ALR
*Brooks, Earl B. ('21).....939 Stuart Bldg., Lincoln, Neb., OALR
*Brooks, E. H.('15).....Zuelke Bldg., Appleton, Wis., OALR

BROOKS, MORRIS J.('34)...455 Ocean Ave., Brooklyn, N. Y., ALR
*BROWN, ALBERT LOUIS ('26)..Doctors Bldg., Cincinnati, O., Op.
*BROWN, AUDREY O.('27)...5057 Woodward, Detroit, Mich., OALR
BROWN, CHARLES WILLIAM('27)
Bank of America Bldg., San Diego, Calif., ALR
BROWN, CLYDE O.('28).3720 Washington Blvd., St. Louis, Mo., ALR
BROWN, CLARENCE J.('34)
Naval Dispensary, Navy Dept., Washington, D. C., ALR
BROWN, C. M. .....510 Delaware Ave., Buffalo, N. Y., ALR
BROWN, JOHN C.('20)..Medical Arts Bldg., St. Paul, Minn., OALR
*BROWN, L. E.('17).....2nd Nat'l Bank Bldg., Akron, O., OALR
BROWN, M. EARLE('33)....921 Canal St., New Orleans, La., Op.
BROWN, MORROW D.('20).....Republic Bldg., Denver, Colo., ALR
BROWN, MORTIMER G.('21)
713 E. Genesee St., Syracuse, N. Y., ALR
BROWN, W. HAROLD('12)
Bank of Montreal Bldg., Edmonton, Alberta, Can., OALR
BROWNELL, MORTON E.('28).105 N. Main St., Wichita, Kan., Op.
BROWNING, ANDREW JOHNSON('35)
Medical Arts Bldg., Portland, Ore., Op.
BROWNING, BIRT LEE('35)..208 Grand St., Newburgh, N. Y., ALR
BRUCE, G. M.('30).....165 W. 165th St., New York, N. Y., Op.
*BRUMBAUGH, JOHN D. ('34)
933 Second Nat'l Bank Bldg., Akron, O., OALR
BRUMM, SETH A.('22).Medical Arts Bldg., Philadelphia, Pa., OALR
BRUNER, ABRAM B.('23).1214 Guardian Bldg., Cleveland, O., OALR
*BRUNO, JOSEPH M. L.('35).31 Jefferson St., Brooklyn, N. Y., Op.
*BRUSEGARD, JAS. F. ('33)....414 Main St., Red Wing, Minn., Op.
BRUST, H. O.('13).....820 S. Crouse Ave., Syracuse, N. Y., ALR
*BRYAN, JAMES LESLIE('28)
919 Bennie-Dillon Bldg., Nashville, Tenn., OALR
*BRYANT, BEN L. ('31).....19 Garfield Place, Cincinnati, O., ALR
BUCHANAN, NORMAN D.('25)
129 Murray St., Peterborough, Ont., Canada, OALR
*BUCHER, WALTER M.('34)..4187 Pearl Road, Cleveland, O., ALR
*BUCK, CLAIR A.('31).....191 Glen St., Glens Falls, N. Y., ALR
BUCKINGHAM, TRACY W.('31).405 Broadway,Bismarck,N.D., ALR
BUCKLEY, J. H.('08)..1st Nat'l Bank Bldg., Ft. Smith, Ark., OALR
BUCKMAN, LEWIS T.('32)
83 S. Franklin St., Wilkes-Barre, Pa., ALR
*BUFFINGTON, WILEY ROSS('35)
Hibernia Bank, New Orelans, La., Op.
BULLARD, CARLETON WM.('32).120 Genesee St.,Auburn,N.Y., ALR
BULLWINKEL, H. GRIFFIN('31)
30 E. 76th St., New York, N. Y., ALR
*BULSON, EUGENE L.('28).406 W. Berry St., Ft. Wayne, Ind., OALR
*BUNNELL, CHESTER WM.('35).121 E. 60th St.,New York,N.Y., ALR
BUONAGURO, MICHAEL J.('31)
589 Lorimer St., Brooklyn, N. Y., Op.
BURDICK, AUSTIN F.('25).330 N.Capitol Ave.,Lansing,Mich., OALR

BURKE, B. RUSSELL('35).....478 Peachtree St., Atlanta, Ga., ALR
BURKE, JOHN R. ('27).....207 Elm St., Holyoke, Mass., OALR
BURLESON, J. H.('17)
1034 Nix Professional Bldg., San Antonio, Tex., OALR
BURMAN, HERMAN J.('35)
1235 Grand Concourse, New York, N. Y., ALR
BURNS, L. J.('30).....1930 Chestnut St., Philadelphia, Pa., ALR
BURNS, S. S.('19)....Missouri Theater Bldg., St. Louis, Mo., ALR
BURRITT, N. W.('30)....30 Beechwood Road, Summit, N. J., ALR
BURTON, FRANK A.('14)..625 Broadway, San Diego, Calif., ALR
*BUSHMAN, L. B.('27).631 City Nat'l Bk. Bldg., Omaha, Neb., OALR
BUSLER, HOWARD S.('35)
N.E. cor. Baltimore and Owen Aves., Lansdowne, Pa., ALR
BUTLER, ROBERT H.('20)
112 W. Columbus Ave., Bellefontaine, O., OALR
BUVINGER, CHARLES W. ('21)
50 Washington St., East Orange, N. J., OALR
BUZZARD, JOSIAH F.('31).....1110 13th Ave., Altoona, Pa., Op.
BYRNES, H. F. .....67 Chestnut St., Springfield, Mass., OALR

C

CADY, D. W.('30)....65 N. Madison Ave., Pasadena, Calif., Op.
*CADY, F. J.('32).....Peoples B. & L. Bldg., Saginaw, Mich., ALR
*CAHILL, HARRY P. ('33)
520 Commonwealth Ave., Boston, Mass., ALR
CALDWELL, ROBT..831 New Donaghey Bldg., Little Rock, Ark., OALR
CALHOUN, JAMES G.('28).Metropolitan Bldg., St. Louis, Mo., Op.
CALLFAS, W. F.....1620 Med. Arts Bldg., Omaha, Neb., ALR
CAMERON, WM. G.('21).Medical Arts Bldg., Tacoma, Wash., OALR
*CAMP, W. E.('20)...Medical Arts Bldg., Minneaplois, Minn., OALR
CAMPBELL, A. A.('30)...86 Bloor St. W., Toronto, Ont., Can., ALR
CAMPBELL, C. H.('18).Maxwell & Bratt Bldg.,Columbus,Neb., OALR
CAMPBELL, DUNCAN A..David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, MALCOLM D.('27).269 Rowena St., Detroit, Mich., OALR
CAMPBELL, ROBERT A..520 LaSalle Bldg., Minneapolis, Minn., OALR
CAMPBELL, W. K.....96 Third Ave., Long Branch, N. J., OALR
CANFIELD, NORTON('33).789 Howard Ave.,New Haven,Conn., ALR
CAPRON, F. P.('14).....118 Angell St., Providence, R. I., OALR
CARDWELL, EDGAR P.('29)....47 Central Ave., Newark, N. J., ALR
CARLETON, RALPH('14)....10 Temple St., Springfield, Mass., Op.
CARMODY, THOMAS J.('26)..408 Temple Bldg., Danville, Ill., OALR
CARNCROSS, HELEN ('20)
R. F. D. No. 1, Carmel-by-the-Sea, Calif., Op.
CARPENTER, CHAPIN('30).1930 Chestnut St.,Philadelphia,Pa., Op.
*CARPENTER, E. W.('22).19½ W. North St., Greenville, S. C., OALR
CARRUTH, HOWARD E.('28)..Med. Arts Bldg., Portland, Ore., OALR
*CARSON, W. E.('17)...Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
CARTER, E. D.('30).....Central Bldg., Wichita, Kan., ALR
CARTER, JOHN M.('29).David Whitney Bldg., Detroit, Mich., ALR
*CARY, E. H. .....Medical Arts Bldg., Dallas, Tex., OALR

CARY, G. C.('18).....Canon Block, Grand Junction, Colo., OALR
*CASSADY, J. VERNAL ('28)
654 Associates Bldg., South Bend, Ind., OALR
CASSIDY, WALDRON A.('31)
1020 Medical Arts Bldg., Omaha, Neb., ALR
*CASTEN, VIRGIL G.('32)
520 Commonwealth Ave., Boston, Mass., Op.
*CASTROVIEJO, RAMON('31).635 W. 165th St., New York,N.Y., Op.
CAVANAUGH, J. A...1712—30 N. Michigan Blvd., Chicago, Ill., ALR
CAVE, LINUS S. ('29).....Physicians' Bldg., Syracuse, N. Y., ALR
CECHA, ALBIN H.('35), U. S. Fleet, Aircraft Battle Force,
U. S. S. Saratoga, San Pedro, Calif., Op.
CHADWICK, IRA B.('20)...Terminal Bldg., Coffeyville, Kan., Op.
*CHAFFERS, WM. H. ('31).....190 Bates St., Lewiston, Me., ALR
CHAINSKI, ED. L.('32).....130 N. Wells St., Chicago, Ill., ALR
*CHAMBERLAIN, WEBB P.('31).7405 Detroit Ave.,Cleveland,O., Op.
*CHANDLER, G. E.('30).....Huntington Bldg., Miami, Fla., ALR
CHAPMAN, S. JEFFERSON('26)
402 Burns Bldg., Colorado Springs, Colo., ALR
CHAPMAN, V. A.('15)
324 E. Wisconsin Ave., Milwaukee, Wis., OALR
CHAPPELL, R. S.('25).Hume Mansur Bldg., Indianapolis, Ind., ALR
CHARLES, JOS.W.('22).3720 Washington Ave., St. Louis, Mo., Op.
CHARLTON, C. COULTER
124 S. Illinois Ave., Atlantic City, N. J., ALR
CHASE, E. FRANK('18).....1008 Cobb Bldg., Seattle, Wash., OALR
CHASE, S. B.('25).....Carver Bldg., Ft. Dodge, Ia., OALR
*CHASE, W. D.('24).....230 E. Broad St., Bethlehem, Pa., ALR
CHATTIN, J. FRANKLIN.....671 Broad St., Newark, N. J., Op.
CHENEY, JAMES W. ....702 Schweiter Bldg., Wichita, Kan., OALR
*CHILDERS, ROBERT J.('29)...604 Park Ave., Plainfield, N. J., ALR
*CHILDS, H. A.('15).....Eagle Block, Creston, Ia., OALR
CHIPMAN, L. D. V.('25).42 Coburg St., St. John, N.B., Can., OALR
CHRISTOPH, CARL H.('28).30 N. Michigan Ave., Chicago, Ill., OALR
*CLARK, CECIL PRATT('31).24 E. Ohio St., Indianapolis, Ind., Op.
CLARK, H. S.('16)..Medical Arts Bldg., Minneapolis, Minn., OALR
*CLARK, I. G. ('20).....188 E. State St., Columbus, O., OALR
CLARK, J. SHELDON('14)..509 State Bk. Bldg., Freeport, Ill., OALR
CLARK, L. H. ('15).....337 Monroe Ave., Rochester, N. Y., OALR
*CLARK, WM. B.('34).American Bank Bldg., New Orleans, La., Op.
CLAY, GRADY E.('17).....Medical Arts Bldg., Atlanta, Ga., Op.
CLEFF, OSCAR('26).....55 E. Washington St., Chicago, Ill., OALR
CLEMENT, C. C. .....30 N. Michigan Blvd., Chicago, Ill., OALR
CLEMENT, L. O.('30).....Guardian Bldg., Salem, Ore., OALR
CLERF, LOUIS HENRY('23).1530 Locust St., Philadelphia, Pa., ALR
CLIFFORD, T. P.('27)
1802 David Whitney Bldg., Detroit, Mich., OALR
CLINE, SAMUEL('32).353 Commonwealth Ave., Boston, Mass., ALR
CLOTHIER, JOSEPH('06).....Kane Bldg., Pocatello, Idaho, OALR
*COATES, GEORGE M.('22)....1721 Pine St., Philadelphia, Pa., ALR

COBB, EDWIN ('12). Masonic Temple Bldg., Marshalltown, Ia., OALR
*CODD, A. N. ('29)
Paulsen Medical and Dental Bldg., Spokane, Wash., ALR
*CODY, CLAUDE CARR ('28)..1304 Walker Ave., Houston, Tex., ALR
COFFELT, THEODORE A. .....Powersite, Mo., OALR
COHEN, HERMAN B. ('27)...255 S. 17th St., Philadelphia, Pa., ALR
*COHEN, NATHAN B. ('35)..232 State St., Perth Amboy, N. J., ALR
COHEN, SAMUEL ('20).....2009 Pine St., Philadelphia, Pa., ALR
COHEN, WM. ('32)...730 Eastern Parkway, Brooklyn, N. Y., Op.
*COHOE, DON A. ('30)..13535 Woodward Ave., Detroit, Mich., ALR
COLBERT, R. M. ('29).....110 W. 55th St., New York, N. Y., ALR
COLGIN, WM. ED. ('35)....605 Columbus Ave., Waco, Texas, Op.
COLLINS, J. D. ('14).....187 Main St., Northampton, Mass., ALR
COLVER, B. N. ('13)....1650 Melwood Drive, Glendale, Calif., ALR
*COMBACKER, LEON C. ('35)
101½ Lincoln Ave., Fergus Falls, Minn., Op.
*COMEE, W. C. ('22).....Northern Bldg., Green Bay, Wis., OALR
*CONBOY, JOHN ED. ('35)
2257 University Ave., New York, N. Y., Op.
CONE, ALFRED J. ('29).....4932 Maryland, St. Louis, Mo., ALR
CONLON, F. A. .....301 Essex St., Lawrence, Mass., OALR
CONNELL, JAS. A. ('33). Citizens Bank Bldg., Riverside Calif., ALR
CONNOLE, J. V. ('32). Miners Nat'l Bk. Bldg., Wilkes-Barre, Pa., Op.
*CONNOR, CHAS. E. ('20)
Lowry Med. Arts Bldg., 4th St. Section, St. Paul, Minn., ALR
*CONSTANS, GEORGE M. ('29)...Clinic Bldg., Bismarck, N. D., Op.
CONSTANTINOPLE, P. S. ('29)
1801 Eye St., N.W., Washington, D. C., ALR
CONWAY, J. M. ('30).....Jenkins Arcade, Pittsburgh, Pa., ALR
*CONWAY, WM. S. ('35)...314½ Howard St., Petosky, Mich., ALR
COOK, W. ALBERT ('24).....Medical Arts Bldg., Tulsa, Okla., OALR
COOMBS, A. J. ('32).....30 N. Michigan Ave., Chicago, Ill., ALR
*COON, EUGENE H. ('35)..131 Fulton Ave., Hempstead, N. Y., ALR
COOPER, C. E. ('17)...652 Metropolitan Bldg., Denver, Colo., ALR
COOPER, ROY C. ('32).....510 S. Aiken Ave., Pittsburgh, Pa., ALR
*COPPS, LYMAN A. ('23)..Marshfield Clinic, Marshfield, Wis., OALR
CORDES, FRED'K C. ('33)...384 Post St., San Francisco, Calif., Op.
*CORSER, J. B. .....345 Wyoming Ave., Scranton, Pa., Op.
*CORTOPASSI, A. J. ('30). 2nd Nat'l Bk. Bldg., Saginaw, Mich., ALR
CORYA, H. W. ('30).....30 E. 40th St., New York, N. Y., ALR
COSTEN, J. B. ('25)..Beaumont Medical Bldg., St. Louis, Mo., ALR
COTT, C. C. .....333 Linwood Ave., Buffalo, N. Y., ALR
COTT, D. N. ('24). 25 Central Ave., St. George, Staten Is., N.Y., ALR
COULTER, CHAS. F.
404 First Nat'l Bank Bldg., Great Falls, Mont., OALR
*COURTNEY, ROBT. H. ('35). Professional Bldg., Richmond, Va., Op.
*COWAN, ALFRED ('28)...1930 Chestnut St., Philadelphia, Pa., Op.
COWAN, CLYDE R. ('33)...Hotel Buckminster, Boston, Mass., ALR
*COWLEY, ROBERT HENRY ('26).....Berea, Ky., OALR
COWPER, H. W. .....543 Franklin St., Buffalo, N. Y., Op.

CRACOVANER, ARTHUR J.('35)
103 E. 78th St., New York, N. Y., ALR
CRAFT, KENNETH L.('20)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
CRAGE, FRANCIS M.('35)..30 N. Michigan Ave., Chicago, Ill., Op.
CRAIG, PAUL C.('33).....220 N. 6th St., Reading, Pa., Op.
CRANE, W. R.('29)....756 S. Broadway, Los Angeles, Calif., ALR
*CRESS, WM. J.('34).....318 W. Market St., Pottsville, Pa., ALR
*CRISP, WM. H.('13).....Metropolitan Bldg., Denver, Colo., Op.
CROCKETT, R. L. ('17).....302 Main St., Oneida, N. Y., Op.
CROISSANT, CHAS. A.('31)..390 Main St., Worcester, Mass., ALR
CROMAN, JOS. M., JR.('24).115 Cass Ave., Mt. Clemens, Mich., OALR
CROSS, A. E. ('14).....390 Main St., Worcester, Mass., Op.
CROSS, G. H.('19).....525 Welsh St., Chester, Pa., Op.
CROSSLEY, E. R.....30 N. Michigan Blvd., Chicago, Ill., OALR
*CROUSHORE, JAS. E.('35).2501 W. Grand Blvd., Detroit, Mich., ALR
CRUME, JOHN J.('31).....Amarillo Bldg., Amarillo, Tex., ALR
*CRUMRINE, NORMAN ROSS('28)...595 Third St., Beaver, Pa., OALR
*CULLER, ARTHUR M.('32)..Fidelity Med. Bldg., Dayton, O., Op.
CULPEPPER, WM. S. ('32)
Station Hosp., Schofield Barracks, T. H., ALR
CUMMINGS, GEO. O.('30).....47 Deering St., Portland, Me., ALR
CUNNING, DANIEL S.('28).145 E. 54th St., New York, N. Y., OALR
CUNNINGHAM, ALLAN R.('24)
260 Barrington St., Halifax, N. S., Canada, OALR
CURDY, R. J.('18).....1102 Grand Ave., Kansas City, Mo., Op.
*CURRAN, E. J. ('19)
1805 Federal Reserve Bank Bldg., Kansas City, Mo., Op.
CURRY, GLENDON E.('12).Westinghouse Bldg., Pittsburgh, Pa., Op.
CURTAIN, EUGENE A.('23)....506 Spruce St., Scranton, Pa., OALR
*CUSHMAN, BEULAH('24).25 E. Washington St., Chicago, Ill., Op.
CUTINO, R. M.('30).....165 Clinton St., Brooklyn, N. Y., Op.
CUTLER, FRANKLIN E. .....Hanna Bldg., Cleveland, Ohio, ALR

D

DAHL, ELMER O.('33)..Med. Arts Bldg., Minneapolis, Minn., ALR
*DAILEY, GILBERT L.('27).....618 N. 3rd St., Harrisburg, Pa., OALR
DAILY, LOUIS('26)....1117 Med. Arts Bldg., Houston, Tex., OALR
*DAILY, RAY K.('26)...1117 Med. Arts Bldg., Houston, Tex., OALR
DANIELS, R. L. .....Box 685, New Bern, N. C., OALR
DANIELSON, RALPH W.('34).Metropolitan Bldg., Denver, Colo., Op.
DARMER, GEO. A.('21).....401 Terminal Bldg., Aurora, Ill., OALR
DARROUGH, L. E.('32).....4105 Live Oak St., Dallas, Tex., ALR
DARROW, C. HOWARD('28)
110 Metropolitan Bldg., Denver, Colo., ALR
*DAVIDSON, H. P. ('24)...30 N. Michigan Ave., Chicago, Ill., Op.
DAVIDSON, MORRIS('29)....80 Centre St., New York, N. Y., Op.
*DAVIES, H. F.('30)...1835 Eye St. N.W., Washington, D. C., ALR
DAVIES, J. S.('27).....2611 N. Union Ave., Tacoma, Wash., OALR
DAVIES, MORGAN C.('32)....269 E. State St., Columbus, O., Op.

DAVIES, T. A.('13).....578 Sherbourne St., Toronto, Can., OALR
DAVIS, CHARLES L.('28)
1305 Medico-Dental Bldg., San Diego, Calif., ALR
DAVIS, DAVID('34)...1835 Eye St. N.W., Washington, D. C., ALR
DAVIS, F. A.('27)...224 W. Washington Ave., Madison, Wis., OALR
DAVIS, G. E. .....1060 Fifth Ave., New York, N. Y., OALR
*DAVIS, HARRY B.('20).....Bryant Bldg., Kansas City, Mo., OALR
DAVIS, J. CALVIN('28).....425 Aquila Court, Omaha, Neb., OALR
DAVIS, J. E.('17).....24½ E. Choctaw, McAlester, Okla., OALR
DAVIS, RALPH F.('20)....Medical Arts Bldg., Portland, Ore., OALR
DAVIS, T. CARROLL('31).3128 N. Broad St., Philadelphia, Pa., ALR
*DAVIS, THOMAS W.('23)
210 O'Hanlon Bldg., Winston-Salem, N. C., OALR
*DAVIS, WARREN B.('34)...135 S. 18th St., Philadelphia, Pa., ALR
*DAVIS, WM. C.('30).....327 E. State St., Columbus, O., Op.
*DAVIS, W. THORNWALL('19)
927 Farragut Sq., Washington, D. C., Op.
*DAY, KENNETH M.('30).121 University Pl., Pittsburgh, Pa., ALR
DAYTON, G. O.....1401 S. Hope St., Los Angeles, Calif., OALR
DEAL, JOHN FRANCIS('28)
First Nat'l Bank Bldg., Springfield, Ill., OALR
*DEAN, ALFRED ('23)
308 Medical Arts Bldg., Grand Rapids, Mich., Op.
*DEAN, L. W. ...Med. School, Washington U., St. Louis, Mo., ALR
*DEAN, WALTER('17).....300 Francis Bldg., Louisville, Ky., OALR
*DEARMIN, ROBERT M. ('31)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
DEBOE, M. P. .....First Nat'l Bank Bldg., Miami, Fla., OALR
DECHERD, H. B.....1515 Medical Arts Bldg., Dallas, Tex., OALR
*DECKER, J. C. ('18).....Frances Bldg., Sioux City, Ia., OALR
DECOSTE, STEPHEN H ('31).1179 Dean St., Brooklyn, N. Y., ALR
*DEFNET, WM. A.('21)..28 Adams Ave. West, Detroit, Mich., ALR
DEICHLER, LYN W.....1930 Chestnut St., Philadelphia, Pa., OALR
*DELANEY, JAMES H.('31).....138 West 9th St., Erie, Pa., Op.
*DELPH, JOHN FRANCIS('28)
104 S. Michigan Blvd., Chicago, Ill., ALR
DENNISON, WARD C.('31).123 Prospect St., Ridgewood, N. J., ALR
DENNIS, EDWARD('21).....309 W. 10th St., Erie, Pa., OALR
*DEPPING, C. W.('30).....19 W. 54th St., New York, N. Y., ALR
DETLING, FRANK E...500 S. Lucas Ave., Los Angeles, Calif., OALR
DEWAR, MURRAY M. ('32)
Medical Arts Bldg., Grand Rapids, Mich., Op.
DEWEESE, CLARENCE..201 West Second St., Lexington, Ky., OALR
DEWEY, C. H.('12), Maj. M. C., U. S. Army,
Letterman General Hospital, San Francisco, Calif., OALR
DEWITT, EDWARD NICHOLAS('28)
881 Lafayette St., Bridgeport, Conn., Op.A
DEXTER, MILTON A.('30)
cor. Pleasant and Prospect Sts., Gloucester, Mass., ALR
DICKERSON, WM. E.('35)....Masonic Temple, Danville, Va., ALR

DICKSON, CHARLES('31).Medical Arts Bldg., Houston, Tex., ALR
DIMMITT, FRANK W.('34).195 Thayer St., Providence, R. I., OALR
*DIMOND, WALDO B.('33)...16 N. Carrol St., Madison, Wis., ALR
*DINTENFASS, HENRY('22).1305 Spruce St., Philadelphia, Pa., ALR
*DIRION, JOSEPHINE KOOB('35)....Rose Bldg., Cleveland, O., Op.
*DIXON, FRED W.('29).....Rose Bldg., Cleveland, O., ALR
DIXON, G. S. .....1150 Fifth Ave., New York, N. Y., R
DIXON, O. JASON('28).Professional Bldg., Kansas City, Mo., ALR
DLUGASCH, LOUIS('29)....110 E. 36th St., New York, N. Y., ALR
DOBBS, O. R.('30).....2743 N. Grand Blvd., St. Louis, Mo., ALR
*DODGE, WARREN M., JR.('34).Sanitarium, Battle Creek, Mich., Op.
DOHERTY, WM. B.('27)..150 W. 55th St., New York, N. Y., Op.
DOLMAN, PERCIVAL('19)..490 Post St., San Francisco, Calif., Op.
DONOHER, WM. D.('17)
1930 Wilshire Blvd., Los Angeles, Calif., OALR
DONOHOE, ED. C.('31)..Professional Bldg., Glendale, Calif., ALR
DORENTE, D. R.('16).....820 S. 25th St., Fort Smith, Ark., OALR
DORGE, RICHARD IRVING('23)
1111 Nicollet Ave., Minneapolis, Minn., OALR
DOUGHERTY, CLIFFORD L.('31)
55 E. Washington St., Chicago, Ill., ALR
DOUGLAS, FREDERICK ALEXANDER('22)
303 State Bank Bldg., La Crosse, Wis., OALR
DOW, FRANK E. ('14).....4 West St., Northampton, Mass., Op.
DOW, JULIAN NEAL('31).Roosevelt Bldg., Los Angeles, Calif., Op.
*DOWLING, HARVEY E. ('32)...Eaton Tower, Detroit, Mich., Op.
DOWLING, J. IVIMEY('14)
116 Washington Ave., Albany, N. Y., OALR
DOWLING, J. THOMAS ('15)
Virginia Mason Hospital Bldg., Seattle, Wash., OALR
*DRISCOLL, F. J.('33).....Reibold Bldg., Dayton, Ohio, ALR
*DUANE, J. F. ('16).....Jefferson Bldg., Peoria, Ill., OALR
DUKE, RONCIE R.('31).....706 Franklin St., Tampa, Fla., ALR
*DULANEY, OCTAVUS('17)...811 Brown Bldg., Louisville, Ky., ALR
DUNCAN, R. A.('29)....339 Amarillo Bldg., Amarillo, Tex., ALR
DUNLAP, LAWRENCE G.('20).101 Main St., Anaconda, Mont., OALR
DUNAWAY, CARL ELLIS('35)..Huntington Bldg., Miami, Fla., ALR
*DUNN, MURISON .....Richmond, Ky., OALR
*DUNNINGTON, J. H.('24)..30 W. 59th St., New York, N. Y., Op.
*DUNPHY, EDWIN B.('31).101 Bay State Road, Boston, Mass., Op.
*DUNTLEY, G. S. ('13)....133 S. Randolph St., Macomb, Ill., OALR
DURR, S. A.('27)....Medico-Dental Bldg., San Diego, Calif., Op.
*DUTROW, H. V.('14)..1040 Fidelity Med. Bldg., Dayton, O., OALR
DYER, B. G.('29).....Santa Fe Hospital, Topeka, Kan., ALR
DYER, C. P.('20).....408 Humboldt Bldg., St. Louis, Mo., Op.

E

*EAGLETON, W. P.('30).....15 Lombardy St., Newark, N. J., ALR
EARLEY, JAMES HUGH('28)
1801 Eye St., N.W., Washington, D. C., ALR

*Earnest, Clarence E.('31) . . . Thatcher Bldg., Pueblo, Colo., ALR
Ebeling, Wm. B.('30) . . . . 54 Greene Ave., Brooklyn, N. Y., Op.
*Eber, Carl Theo.('28) . 1006 Carleton Bldg., St. Louis, Mo., Op.
Ebert, Edwin C. ('29)
U. S. Navy, Navy Dept., San Diego, Calif., ALR
Edgerton, Ambrose Earl('31)
450 Sutter St., San Francisco, Calif., Op.
Edson, R. A.('14) . . . . . 957 Delaware Ave., Buffalo, N. Y., Op.
*Edwards, C. J.('13) . . 1st Nat'l Bank Bldg., Vicksburg, Miss., OALR
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Elles, Norma B.('19)
1704 Niels Esperson Bldg., Houston, Tex., OALR
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Ellis, Z. H. ('27) . . . . . 58 E. 65th St., New York, N. Y., Op.
*Embery, Frank ('27)
4801 Penn St., (Frankford), Philadelphia, Pa., ALR
*Engler, Clarence Wm.('33)
2323 Prospect Ave., Cleveland, Ohio, ALR
Enlows, Ella M. A.('34) . 1726 Eye St. N.W., Washington, D.C., ALR
Equen, Murdock S. ('31)
384 Peachtree St. N.W., Atlanta, Ga., ALR
Ersner, Matthew S. . . . . 1915 Spruce St., Philadelphia, Pa., ALR
Estabrook, Chas. T. . . 10 Algonquin Road, Worcester, Mass., OALR
*Eubank, Ambrose E. ('28)
636 Argyle Bldg., Kansas City, Mo., OALR
Evans, J. N.('25) . . . . 23 Schermerhorn St., Brooklyn, N. Y., Op.
Evans, Maurice George('32)
311 Commonwealth Ave., Boston, Mass., ALR
Evans, S. S.('32) . Phys- & Surgeons Bldg., Memphis, Tenn., ALR
*Evans, Wm. Hiram('34) . Dollar Bank Bldg., Youngstown, O., ALR
Eves, C. C. ('32) . . . . . 1910 Spruce St., Philadelphia, Pa., ALR
Ezell, H. C.('26) . . . . . 228 Doctors Bldg., Nashville, Tenn., OALR

F

*Fackler, C. L.('30) . . . . . 11 E. Market St., York, Pa., ALR
Faier, Samuel Z.('35) . . . Medical Arts Bldg., Omaha, Nebr., ALR
Fairbairn, John F. . . . . 925 Delaware Ave., Buffalo, N. Y., ALR
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Fallows, Howard D.('18) . Foresters Bldg., Mason City, Ia., OALR
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Farnsler, H. Hershey . . . . 1438 Market St., Harrisburg, Pa., OALR
Farnsworth, M. A.('17) . . . McNeen Bldg., La Junta, Colo., OALR
Farrell, Jas. I.('35) . . . . . 250 Genesee St., Utica, N. Y., Op.
*Fawcett, Ivan ('13) . . . . . 75 Twelfth St., Wheeling, W. Va., OALR

FELDMAN, LOUIS A.('35) .259 New York Ave., Brooklyn,N.Y., Op.
*FELLOWS, M. FORDING('35) . .Med. Arts Bldg., Duluth, Minn., Op.
FEASTER, HENRY J. ('31) . . . . .461 77th St., Brooklyn, N. Y., ALR
*FENTON, RALPH A.('21) .806 Med. Arts Bldg., Portland, Ore., OALR
*FERGUSON, E. R.('35) . .P. and S. Hospital, Cleveland, Tenn., ALR
FERGUSON, R. J.('27) . . . .59 College St., New Haven, Conn., OALR
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8577 112th St., Richmond Hill, New York, N. Y., ALR
FERRIS, DAVID P.('28) .Missouri Thea. Bldg., St. Louis, Mo., ALR
*FERRIS, RALPH G. ('34)
55 W. Maple Ave., Birmingham, Mich., ALR
FEWELL, A. G. ('31) . . . . . .1924 Pine St., Philadelphia, Pa., Op.
FIELDING, F. G. ('25) . . . . . .70 Ridge St., Glens Falls, N. Y., ALR
FIGI, F. A. ('27) . . . . . .Mayo Clinic, Rochester, Minn., LR
FINDLAY, E. K. ('30) . . . . .30 N. Michigan Ave., Chicago, Ill., Op.
FINE, ABRAHAM ('32) . . .1505 Ocean Ave., Brooklyn, N. Y., Op.
*FINEBERG, MAXWELL ('30)
Missouri Theatre Bldg., St. Louis, Mo., ALR
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*FISCHBACH, V. W.('31)
2600 Union Central Bldg., Cincinnati, O., ALR
FISCHER, AUBREY ('34)
1835 Eye St. N.W., Washington, D. C., ALR
FISCHER, N. ARTHUR('26)
623 Union Trust Bldg., Pittsburgh, Pa., ALR
FISCHER, V. B.('17) .203 Mercantile Bk. Bldg., Boulder, Colo., OALR
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Jersey Central Bldg., Asbury Park, N. J., OALR
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FISKE, GEORGE F. . . . . .25 E. Washington St., Chicago, Ill., OALR
FITZGERALD, FRED J. C. ('34)
424 Madison Ave., New York, N. Y., Op.
FITZGERALD, J. P.('25) . . .30 N. Michigan Blvd., Chicago, Ill., Op.
FLATLEY, T. J.('14) . . . . . .Reliance Bldg., Moline, Ill., OALR
FLOYD, T. W.('18) . . . . . .811 Peoria Life Bldg., Peoria, Ill., OALR
FLYNN, CHAS. T.('27) . .41 Thumbull St., New Haven, Conn., ALR
*FLYNN, JAMES A.('24)
1511 Rhode Island Ave., N.W., Washington, D. C., ALR
FOGARTY, CHAS. W.('20) . .372 St. Peter St., St. Paul, Minn., OALR
*FOLK, MAX LYON('28) . .25 E. Washington St., Chicago, Ill., Op.
FOLKEN, FRANK GARRETT ('28)
Freeborn County Nat'l Bank Bldg., Albert Lea, Minn., OALR
*FOLSOM, W. H.('27) . . . .11 N. Main St., Fond du Lac, Wis., OALR
FONT, JUAN H.('33) .Cruz St. No. 13, San Juan, Puerto Rico, ALR
FORBES, SHERMAN B.('22) . . . .706 Franklin St., Tampa, Fla., OALR

*Ford, Hanby Lewis('28) .401 Robeson Bldg., Champaign, Ill., OALR
*Forgrave, L. Robert('23) . .204 Logan Bldg., St. Joseph, Mo., OALR
Forsythe, S. T.('19) . . . . . Rose Bldg., Cleveland, O., OALR
*Fort, A. G. ('19) . . . . . Doctors Bldg., Atlanta, Ga., OALR
*Foster, Harold('35)
9205—35th Ave., Jackson Heights, New York, N. Y., ALR
Foster, J. H.('13) . . . . . 1304 Walker Ave., Houston, Texas, ALR
*Foster, Warren H.('35) . . . . . 217 Fifth Ave. S., Clinton, Ia., ALR
*Foster, Wayne Jones('35) . . Higley Bldg., Cedar Rapids, Ia., ALR
Fouts, John D.('20) . . . . . Fidelity Bldg., Dayton, O., OALR
Fowler, Earl B.('25) . . . 55 E. Washington St., Chicago, Ill., Op.
Fowler, Robert H.('29) .149 E. 62nd St., New York, N. Y., ALR
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Fowler, Wm.('25) . . . . . Maccabee Bldg., Detroit, Mich., OALR
Fowlkes, John W.('29) . .30 W. 59th St., New York, N. Y., ALR
*Fox, C. H.('20) . . . . . 5 W. 21st St., Kearney, Nebr., OALR
Fox, Noah('26) . . . . . .841 E. 63rd St., Chicago, Ill., ALR
*Fralick, F. Bruce('33)
First Nat'l Bank Bldg., Ann Arbor, Mich., Op.
Frankel, Edward, Jr.('28) .217 E. 17th St., New York, N. Y., ALR
*Fraser, R. H.('25)
Security Nat'l Bank Bldg., Battle Creek, Mich., ALR
Fraser, Robert C.('29) .940 Military St., Port Huron, Mich., ALR
Fraser, W. G.('27) . . . .Medical Arts Bldg., Ottawa, Canada, OALR
*Freedman, Abraham O.('32)
1396 St. Catherine St., W., Montreal, Canada, ALR
Freedman, L. M. ('20)
475 Commonwealth Ave., Boston, Mass., ALR
Friedman, Benj.('33) . . . . .6 W. 77th St., New York, N. Y., Op.
Freimuth, L. E.('27) .University Club Bldg., St. Louis, Mo., ALR
*French, R. F.('13) . . . . .Masonic Temple, Marshalltown, Ia., OALR
Frey, W. G., Jr.('34) . . . . . 121 E. 60th St., New York, N. Y., Op.
Friedenwald, J. S.('25) . . . .1212 Eutaw Pl., Baltimore, Md., Op.
Friedman, Benjamin('35) .6 W. 77th St., New York, N. Y., Op.
*Frost, Albert Dalbey('28) .681 E. Broad St., Columbus, O., Op.
Frothingham, Edward('28) .8644 94th St.,Woodhaven,N.Y., OALR
Fry, W. E.('31) . . . . . 1912 Spruce St., Philadelphia, Pa., Op.
Fuchs, Valentine Henry('28)
American Bank Bldg., New Orleans, La., ALR
Fulkerson, C. B.('14)
Room 403, 107 W. Michigan Ave., Kalamazoo, Mich., OALR
Fullenwider, C. M. . . . . Barnes Bldg., Muskogee, Okla., OALR
Fuller, Harry B.('26) . . . .2219 E. 70th Place, Chicago, Ill., OALR
Fuller, John Andre('28) . .1st Nat'l Bank Bldg., Reno, Nev., OALR
Fuller, T. E.('15) . . . . . 209 Pine St., Texarkana, Tex., OALR
Furgason, A. P.('17) . . . . . Richards Blk., Lincoln, Neb., OALR
Furlong, Thos. F., Jr.('35) .Times Med. Bldg.,Ardmore,Pa., ALR
*Furstenberg, A. C.('25) .1st Nat'l Bldg., Ann Arbor, Mich., ALR
*Fust, John H. E. ('22) . . . . . 231 West 7th St., Erie, Pa., OALR

G

GABRIEL, C. K.('30)...Illinois State Bank Bldg., Quincy, Ill., ALR
GADBOIS, ARTHUR E.('21)
DeLay Nat'l Bank Bldg., Norfolk, Neb., OALR
*GAILEY, WATSON W.('22).Griesheim Bldg., Bloomington, Ill., OALR
GALE, M. JEAN('18).....Republic Bldg., Denver, Colo., Op.
GALLAGHER, JOHN H. C.('27).105 Center St., Chicopee, Mass., ALR
*GALLOWAY, T. C.('27).....636 Church St., Evanston, Ill., ALR
*GAMBLE, R. C.('29).....30 N. Michigan Ave., Chicago, Ill., Op.
GARLAND, FREDERICK E.('34).483 Beacon St., Boston, Mass., LR
GARRAGAN, EDW. F.('08).25 E. Washington St., Chicago, Ill., OALR
GARRETT, ERNEST W.('35).....Hanna Bldg., Cleveland, O., ALR
*GARRETT, JOHN D.('32)..Bar Ass'n Bldg., Indianapolis, Ind., ALR
GATEWOOD, E. T.('25)...Professional Bldg., Richmond, Va., ALR
GATEWOOD, W. L.('15).....330 Park Ave., New York, N. Y., ALR
GAUDET, LUCIEN S.('28)...405 Franklin St., Natchez, Miss., OALR
GAUDREAU, HONORE E.('34).293 Bridge St.,Springfield,Mass., Op.
GEIGER, A. H.('29).....554 Fullerton Parkway, Chicago, Ill., ALR
GEMEROY, JOS. C.('35)...Henry Ford Hosp., Detroit, Mich., ALR
GEORGE, HENRY W.('18)..19 N. Union St., Middletown, Pa., Op.
*GERSTLEY, MANFRED J.('33).30 E. 40th St., New York, N. Y., ALR
*GESSLER, WM. F.('33)..2902 Fairfield Ave., Ft. Wayne, Ind., Op.
GIBBY, HAROLD J. ('31).....27 Elm St., Worcester, Mass., ALR
*GIBSON, GLEN G.('35).....Beury Bldg., Philadelphia, Pa., Op.
GIFFORD, S. R.('19).....58 E. Washington St., Chicago, Ill., Op.
GILBERT, FRANK Y.....148 Park St., Portland, Me., OALR
GILL, ARTHUR E.('28)..Hyrum, Utah, and El Centro, Calif., OALR
GILL, E. G.('35).....711 S. Jefferson St., Roanoke, Va., ALR
GILL, G. BACHE('23).1726 Eye St. N.W., Washington, D. C., ALR
*GILL, WM. DAVIS('29).705 E. Houston St.,San Antonio,Tex., OALR
GILLETT, WILBUR G.('20)..201½ N. Main St., Wichita, Kan., Op.
*GILLETTE, D. F.('25)...State Tower Bldg., Syracuse, N. Y., Op.
GILLILAND, O. S.('30)..Professional Bldg., Kansas City, Mo., ALR
GILLUM, J. R.('20).....221 S. 6th St., Terre Haute, Ind., OALR
*GIPNER, J. F.('26)....Medical Arts Bldg., Rochester, N. Y., Op.
*GITTENS, T. R. ('20)....401 Davidson Bldg., Sioux City, Ia., OALR
GLASFORD, S. T.('24).....333 Court St., Pekin, Ill., OALR
GLATT, MORRIS A. .....310 S. Michigan Ave., Chicago, Ill., ALR
*GLICK, HARRY N.('28)..3115 S. Grand Blvd., St. Louis, Mo., ALR
*GLOSSER, HERBERT H.('12).448 Franklin St., Buffalo, N. Y., Op.
*GLOVER, ARTHUR C.('33).311 W. Church St., Elmira, N. Y., OALR
GLOVER, S. P.('17).....1118 Twelfth Ave., Altoona, Pa., OALR
GLOWACKI, BEN FRANCIS('28)
770 Maccabee Bldg., Detroit, Mich., OALR
*GOAD, R. R.('31).....Laurel Bldg., Muscatine, Iowa, Op.
GOAR, EVERETT LOGAN('26).1300 Walker Ave., Houston, Tex., Op.
GOLDBERG, SYLVAN S.('35)
1917 Wilshire Blvd., Los Angeles, Calif., ALR
*GOLDCAMP, ED. C.('32)..Dollar Bank Bldg., Youngstown, O., ALR
GOLDEN, MATTHEW G.('33)...1 Nevens St., Brooklyn, N. Y., ALR

GOLDENBURG, MICHAEL ('31)
104 S. Michigan Ave., Chicago, Ill., Op.
*GOLDING, JOSEPH E. ('25) . . 170 Hancock St., Brooklyn, N. Y., Op.
GOLDMAN, A. MILTON ('28) . 121 E. 60th St., New York, N. Y., Op.
GOLDMAN, H. G. ('23) . . . . . 57 W. 58th St., New York, N. Y., ALR
*GOLDSMITH, PERRY G. ('12) . Med. Arts Bldg., Toronto, Can., ALR
GOLDSTEIN, ISIDORE ('29) . . 1095 Park Ave., New York, N. Y., Op.
GOLDTHWAITE, RALPH H. ('35)
Gorgas Hospital, Canal Zone, Panama, OALR
*GOOCH, JOHN B. ('35) . . . Canal Bank Bldg., New Orelans, La., ALR
*GOOD, WM. M. ('29) . . . . . 63 Center St., Waterbury, Conn., ALR
GOODALL, EDWIN B. ('34) . 101 Bay State Road, Boston, Mass., Op.
GOODE, ED. A. ('29) . . . 143-28 Sanford Ave., Flushing, N. Y., ALR
GOODELL, WM. ('34) . . . . 121 Chestnut St., Springfield, Mass., ALR
GOODMAN, THOMAS L. ('31) . Med. Arts Bldg., Ft. Worth, Tex., ALR
GOODSPEED, HARRY B. ('34) . 36 Pleasant St., Worcester, Mass., ALR
*GOODYEAR, H. M. ('24) . . . . . Doctors Bldg., Cincinnati, O., ALR
GORDON, CHAS. H. ('26) . . . . . 46 Deering St., Portland, Me., ALR
GORDON, GLENN G. ('29) . . . . . Alexander Bldg., Baker, Ore., ALR
GORDON, J. S. ('19) . . . . 922 Bankers Bldg., Milwaukee, Wis., OALR
GORDON, WILLIAM ('33) . . . . . 1721 Pine St., Philadelphia, Pa., ALR
GORRELL, T. J. H.
16th and Vincennes Ave., Chicago Heights, Ill., OALR
GOSNEY, C. W. ('20) . . . . . Taft Bldg., Hollywood, Calif., Op.
GOTTLIEB, MARK I. . . . . 2 West 87th St., New York, N. Y., ALR
GOUGH, ROY H. ('31) . Medical Arts Bldg., Fort Worth, Tex., OALR
*GOURLEY, G. F. ('17) . . . . . Sinclair Bldg., Steubenville, O., OALR
GOUTERMAN, J. I. ('30) . . 1930 Chestnut St., Philadelphia, Pa., Op.
*GOUX, RAYMOND SALOT ('22)
545 David Whitney Bldg., Detroit, Mich., OALR
GOWEN, FRANCIS V. ('29)
904 E. Chelton Ave., Philadelphia, Pa., ALR
GOWEN, GEO. R. ('29) . 306 Baker Bldg., Walla Walla, Wash., ALR
*GRACE, CHAS. C. ('33) . . East Coast Hosp., St. Augustine, Fla., ALR
*GRACEY, GEO. F. ('24) . . . . . 209 State St., Harrisburg, Pa., OALR
GRAEBNER, HERBERT ('34) . . 45 Park Ave., New York, N. Y., ALR
GRANBERRY, CARL ('34)
1124 Maison Blanche Bldg., New Orleans, La., ALR
GRANGER, W. B. ('20) . . . . . Gazette Bldg., Emporia, Kan., OALR
GRANT, FRED E. ('12)
Kalamazoo Nat'l Bank Bldg., Kalamazoo, Mich., OALR
GRANT, H. W. ('30) . . . . . Lowry Bldg., St. Paul, Minn., Op.
GRANT, LEE E. ('27) . . . David Whitney Bldg., Detroit, Mich., OALR
GRAY, E. B. ('27) . 909 Montgomery Bldg., Spartanburg, S. C., ALR
*GREEAR, J. N., JR. ('29) . 1740 M St. N.W., Washington, D. C., Op.
GREEN, A. S. ('18)
The Green's Eye Hosp., 1801 Bush St., San Francisco, Calif., Op.
GREEN, HYMAN S. ('35) . . . . . 1005 Belmont Ave., Chicago, Ill., ALR
GREEN, L. D. ('18)
The Green's Eye Hosp., 1801 Bush St., San Francisco, Calif., Op.

*GREENE, JOSEPH BERRY('23).Haywood Bldg.,Asheville,N.C., ALR
GREENE, LAURENCE W.('28)..Republic Bldg., Denver, Colo., ALR
GREENE, M. L.('30)..Missouri Theatre Bldg., St. Louis, Mo., Op.
GREENFIELD, SAMUEL D. ('27)
169 New York Ave., Brooklyn, N. Y., ALR
GREENFIELD, WILLIAM JOHN('28)
50 Anderson St., Hackensack, N. J., ALR
*GREENWOOD, G. J. ('30)..104 S. Michigan Ave., Chicago, Ill., ALR
636 Church St., Evanston, Ill.
GREEVER, B. L.('20).....100 N. Main St., Hutchinson, Kan., OALR
GREGG, JOHN B.('20)
612 Security Nat'l Bank Bldg., Sioux Falls, S. D., OALR
GRESSER, ED B.('29).....816 Fifth Ave., New York, N. Y., Op.
GRIFFIN, EDWIN A.('34)..165 Hancock St., Brooklyn, N. Y., ALR
*GRIFFEY, ED. W.('29)....Medical Arts Bldg., Houston, Tex., ALR
GRIFFITH, H. M.('30).Johnstown Trust Bldg.,Johnstown,Pa., Op.
GRIM, U. J.....30 N. Michigan Blvd., Chicago, Ill., OALR
GRIMES, C. H.('32)....3546 N. Broad St., Philadelphia, Pa., ALR
GRISCOM, J. MILTON.....255 S. 17th St., Philadelphia, Pa., Op.
GRISWOLD, GEO. W.('35).....211 W. 3rd St., Roswell, N. M., ALR
GROSS, JULIUS E.('20).....3401 Fifth Ave., Pittsburgh, Pa., ALR
GROSVENOR, L. N.('07)...366 Dakota Ave. S., Huron, S. D., OALR
*GROVE, W. E.('14)..324 E. Wisconsin Ave., Milwaukee, Wis., OALR
GSELL, J. F.('20).....911 Beacon Bldg., Wichita, Kan., OALR
GUERINOT, A. J.('18).604 Westinghouse Bldg.,Pittsburgh,Pa., OALR
*GUGGENHEIM, L. K. .....6227 Rosebury, St. Louis, Mo., Op.
*GUIBOR, GEO. P.('33).....Central Life Bldg., Ottawa, Ill., Op.
and 30 N. Michigan Ave., Chicago, Ill.,
GUNDELACH, C. A..825 University Club Bldg., St. Louis, Mo., ALR
*GUNDERSEN, TRYGVE('35)..5 Bay State Road, Boston, Mass., Op.
*GUNN, M. L. ('29).....Smith Bldg., Harlan, Ky., ALR
*GUTTMAN, M. REESE('29).185 N. Wabash Ave., Chicago, Ill., ALR
GUY, LOREN P.('34).....116 E. 62nd St., New York, N. Y., Op.
*GUYTON, B. S.('18).....512 Van Buren Ave., Oxford, Miss., OALR

H

*HACKNEY, F. J.('13).311 McCallie Ave., Chattanooga, Tenn., OALR
HADEN, HENRY C. .....1914 Travis St., Houston, Texas, Op.
HAESSLER, F. HERBERT('29)...Wells Bldg., Milwaukee, Wis., Op.
HAGAMAN, VAN DYKE('34).Lamar Life Bldg, Jackson, Miss., ALR
HAGEMANN, JOHN A.('25)..Highland Bldg., Pittsburgh, Pa., ALR
HAGLER, E. E.('96)....401 E. Capitol Ave., Springfield, Ill., OALR
HAHN, WM. H.('30).....15 Lombardy St., Newark, N. J., Op.
*HAIRE, PAUL G.('35).....6 Pleasant St., Malden, Mass., Op.
*HALE, ARTHUR S. ('28)...1609 Eaton Tower, Detroit, Mich., OALR
HALEY, WM. T.('33).....531 Beacon St., Boston, Mass., ALR
HALL, ALICE K.('24).....25 E. Washington St., Chicago, Ill., ALR
*HALL, EDWIN PERRY('28)...76 Chestnut St., Oneonta, N. Y., ALR
*HALL, SOBISCA S.('34).95 Fairmont Ave., Fairmont, W. Va., ALR

HALLETT, G. DEWAYNE ('27)
274 W. 86th St., New York, N. Y., Op.
*HALLUM, ALTON V.('34) . . . .478 Peachtree St., Atlanta, Ga., Op.
HALPER, PHILIP A.('32) .307 N. Michigan Ave., Chicago, Ill., OALR
HALSTED, FRED S.('18) .738 Metropolitan Bldg.,Denver,Colo., OALR
*HAMLIN, FRED EUGENE('26) .Med. Arts Bldg., Roanoke, Va., ALR
*HAMMOND, ARTHUR('35) .D. Whitney Bldg., Detroit, Mich., ALR
*HAMPSEY, A. R. ('30) . . . . . .May Bldg., Pittsburgh, Pa., ALR
HAMPTON, ROBT. R. . .804 Boston Bldg., Salt Lake City, Utah, ALR
HAND, FRANK('35) . . . . . .490 Post St., San Francisco, Calif., ALR
HANDS, SIDNEY GILLOTT('26) .201 W. 2nd St., Davenport, Ia., ALR
HANEY, WM. P.('20) .1500 Medical Arts Bldg., Omaha, Nebr., OALR
*HANS, C. L.('21) . . . . . .Provident Bank Bldg., Cincinnati, O., OALR
*HANSEL, FRENCH K.('26) .634 N. Grand Ave., St. Louis, Mo., ALR
*HANSEN, ERLING W.('20) .Med. Arts Bldg.,Minneapolis,Minn., OALR
HANSON, WM. L.('29) . .707 Spivey Bldg., East St. Louis, Ill., ALR
*HARA, H. JAS.('33) . . .432 S. Boyle Ave., Los Angeles, Calif., ALR
HARALSON, CHAS. H.('29) .816 Med. Arts Bldg., Tulsa, Okla., Op.
*HARBERT, JOHN P.('18) . .135 N. Main St., Bellefontaine, O., OALR
HARDESTY, J. F.('22) . .Missouri Theatre Bldg., St.Louis,Mo., Op.
HARDIE, G. C.('30) . . .290 W. Michigan Ave., Jackson, Miss., ALR
HARDY, LEGRAND H.('28) . . .30 E. 40th St., New York, N. Y., Op.
HARDY, W. F.('14) . . . . . .Humboldt Bldg., St. Louis, Mo., Op.
HARGITT, CHAS. A.('26) .17 Schermerhorn St., Brooklyn,N.Y., Op.
HARRELL, VOSS('26) . . . . . .Hofman Bldg., Detroit, Mich., ALR
*HARRIS, ED. W.('30) . . . . . .681 E. Broad St., Columbus, O., ALR
HARRIS, J. HARLEY('31) . .Med. Arts Bldg., Memphis, Tenn., ALR
HARRIS, ROBIN('23) . . .500 Lamar Life Bldg., Jackson, Miss., ALR
HARRIS, THOMAS J.('29) . . .104 E. 40th St., New York, N. Y., ALR
HARRISON, FRED HAZARD('28)
308 First Nat'l Bank Bldg., Connellsville, Pa., OALR
*HARTLEY, F. A. . . . . .302 E. High St., Springfield, O., OALR
*HARTMAN, EUGENE C. ('33)
610½ Market St., Parkersburg, W. Va., OALR
HARTSHORNE, ISAAC('26) . .30 W. 59th St., New York, N. Y., Op.
Also 22 Commonwealth Ave., Boston, Mass.,
HARTZELL, SOL. M. . . . .Dollar Bank Bldg., Youngstown, O., OALR
HASELTINE, BURTON('27) .122 S. Michigan Ave., Chicago, Ill., OALR
HASKELL, ALFRED W. . . . . .142 High St., Portland, Me., Op.
HASKIN, W. H. ('13) . . . . . .300 Park Ave., New York,N.Y., ALR
HASPEL, M. DAVID('11)
Maison Blanche Bldg., New Orleans, La., OALR
HASTY, F. E.('25) . . . .Medical Arts Bldg., Nashville, Tenn., ALR
HAUER, A. M. ('16) . . . . . .327 E. State St., Columbus, O., ALR
HAWMAN, ERLE G.('20) .4051 N. Broad St., Philadelphia, Pa., Op.
HAYS, HAROLD M.('12) . . .133 E. 58th St., New York, N. Y., ALR
HAZLETT, L. R.('13) . . .Savings and Trust Bldg., Butler, Pa., OALR
*HEATH, PARKER('24) . .David Whitney Bldg., Detroit, Mich., Op.
HEBERT, ARTHUR W. . .450 Sutter St., San Francisco, Calif., OALR
HEDRICK, HAROLD B.('29) .Professional Bldg.,Kansas City,Mo., ALR

HEEB, HARRY J. . .204 W. Wisconsin Ave., Milwaukee, Wis., OALR
HEED, C. R. . . . . .1205 Spruce St., Philadelphia, Pa., Op.
*HEETDERKS, D. R.('30) . Med. Arts Bldg., Grand Rapids, Mich., ALR
HEINBERG, CHAS. J.('31) . . . . . .Blount Bldg., Pensacola, Fla., ALR
*HELFRICH, ED D.('30) . . . . . .269 E. State St., Columbus, O., ALR
HEMPSTEAD, BERT E.('20) . . . . Mayo Clinic, Rochester, Minn., ALR
HENDERSON, L. E.('30) . . .168 Sterling St., Watertown, N. Y., Op.
*HENDRICKS, LOUIS J.('31) . . .210 W. 12th St., Cincinnati, O., Op.
HENNESSEY, WM. W. . . . . .333 Essex St., Salem, Mass., OALR
HENNINGER, L. L.('13) .65 N. Madison Ave., Pasadena, Calif., OALR
HENTON, G. E.('18) .720 S.W. Washington St., Portland, Ore., OALR
HERRIMAN, L. L.('18) . First State Bank Bldg., Alamosa, Calif., OALR
HETRICK, L. E.('23) . . . . . .40 E. 61st St., New York, N. Y., ALR
And 260 North Ave., New Rochelle, N. Y.
*HEWSON, WM.('31)
6013 Greene St., Germantown, Philadelphia, Pa., ALR
*HICKEY, H. L.('30) . . . . . .Republic Bldg., Denver, Colo., ALR
*HICKMAN, WARREN R.('34) .211 Third St., Logansport, Ind., ALR
*HICKS, VONNIE M.('26) . .Professional Bldg., Raleigh, N. C., Op.
HICKS, WM. J.('30) . . . . . .15 King St., Middletown, N. Y., ALR
HIGBEE, D. R.('27) . . . .3245 Fourth Ave., San Diego, Calif., ALR
HIGGINS, R. P.('24) . . . . . .20 Court St., Cortland, N. Y., OALR
*HIGGINS, S. G. . . .324 E. Wisconsin Ave., Milwaukee, Wis., OALR
HIGH, CLIFTON E.('35) . . . . .Small Bldg., Wellington, Texas, Op.
HIGH, ISAAC B.('31) . . . . . .326 N. 5th St., Reading, Pa., ALR
HILDING, ANDERSON C.('31) . Med. Arts Bldg., Duluth, Minn., ALR
*HILDRETH, H. ROMMEL ('34)
824 Metropolitan Bldg., St. Louis, Mo., Op.
*HILGARTNER, H. L.('30) . . . . . .Norwood Bldg., Austin, Tex., Op.
HILGARTNER, HENRY L., JR.('35) . Norwood Bldg., Austin, Tex., Op.
HILL, C. E.('19) . . . . . .160 Bloor St., W., Toronto, Can., Op.
HILL, EMORY('16) . . . . . .501 E. Franklin St., Richmond, Va., Op.
*HILL, FRED'K THAYER('28) . Professional Bldg., Waterville, Me., ALR
HILL, HOWARD F.('28) . . . . Professional Bldg., Waterville, Me., Op.
*HINMAN, E. E.('24) . . .146 Washington Ave., Albany, N. Y., ALR
*HIRSCH, CAESAR('34) . .667 Madison Ave., New York, N. Y., ALR
*HIRST, OTTO C. ('32) . . . . .255 S. 17th St., Philadelphia, Pa., ALR
*HITZ, JOHN B.('34) . 208 E. Wisconsin Ave., Milwaukee, Wis., Op.
HOBART, CARL('29) . . . . . .Metropolitan Bldg., St. Louis, Mo., Op.
*HOCHFILZER, JOHN J.('35)
Lowry Med. Arts Bldg., St. Paul, Minn., ALR
HODGE, GEO. E.('30) . . .1414 Drummond St., Montreal, Can., ALR
*HODSON, B. F. . . . . .159 N. E. 29th St., Miami, Fla., OALR
HOFFMANN, JOHN N.('27) . . . . .309 Brant Bldg., Canton, O., ALR
HOFFMAN, L. G.('27) . . . . . .30 N. Michigan Ave., Chicago, Ill., OALR
HOFFMAN, WALTER F.('20) .1317 Marion St., Seattle, Wash., Op.
HOFFMAN, WM. A.('32) .231 W. Washington St., Chicago, Ill., Op.
*HOFLING, CHAS. A.('29) . . . . . .707 Race St., Cincinnati, O., Op.
*HOFMANN, W. P.('25) . . . . . .707 Kahl Bldg., Davenport, Ia., OALR
HOGAN, W. L.('24) . . . . . .750 Main St., Hartford, Conn., OALR

*Hogshead, J. M. .....602 Georgia Ave., Chattanooga, Tenn., OALR
Hohf, Julius A.('15).....Clinic Bldg., Yankton, S. D., OALR
*Holland, Emory Ed.('33)....51 S. 8th St., Richmond, Ind., Op.
*Hollander, Abraham R.('31)
30 N. Michigan Ave., Chicago, Ill., ALR
*Holloway, J. M.('32)
N.W. cor. Hanover and Charles Sts., Fredericksburg, Va., ALR
Holloway, Thos. B.....1819 Chestnut St., Philadelphia, Pa., Op.
Holmes, Benjamin H.('22).....523 Main St., Racine, Wis., ALR
Holmes, Will W.('35)..Masonic Temple, Logansport, Ind., ALR
*Holzberger, John C.('32)
7005 Freshpond St., Brooklyn, N. Y., ALR
Holzer, Wm. F. ('31)...36 Pleasant St., Worcester, Mass., Op.
*Hompes, J. J. .....702 Sharp Bldg., Lincoln, Neb., OALR
*Hoople, Gordon D.('29)..Med. Arts Bldg., Syracuse, N. Y., ALR
Hooss, C. H. A.('30)....3115 S. Grand Blvd., St. Louis, Mo., Op.
Hoover, Walter Boyd ('34)
605 Commonwealth Ave., Boston, Mass., ALR
Hopkins, G. H.('30).....702 N. Main St., Pueblo, Colo., Op.
Horn, W. L.('29).....1040 Park Ave., New York, N. Y., ALR
*Hornback, Geo. A.('35).....500 Broadway, Hannibal, Mo., ALR
Horner, Warren D.('35).384 Post St., San Francisco, Calif., Op.
*Hotchkiss, Walter T.('31)
541 Lincoln Road, Miami Beach, Fla., ALR
Hourn, George E.('20)
513 University Club Bldg., St. Louis, Mo., ALR
Houser, Karl M.('34)...2010 Spruce St., Philadelphia, Pa., ALR
Hovde, A. G.('16).....Taft Bldg., Hollywood, Calif., OALR
Howard, C. Norman('19)....Warsaw Clinic, Warsaw, Ind., OALR
Howard, Edward L.('26).405 Med. Arts Bldg.,Ft.Worth,Tex., OALR
Howard, Harvey J.('28)..Park Plaza Hotel, St. Louis, Mo., Op.
Howard, J. W.....Professional Bldg., Kansas City, Mo., OALR
Howard, Lloyd G.('23).Co. Bluffs Clinic, Council Bluffs, Ia., OALR
*Howard, Robt. E.('31).2600 Union Cent. Bldg.,Cincinnati,O., ALR
Howard, W. H.('23).Phys.&Surg. Bldg., Minneapolis, Minn., OALR
Howe, A. W.('22).....Medical Arts Bldg., Tacoma, Wash., OALR
Howe, Spencer S.('29)
419 Bellingham Nat'l Bank Bldg., Bellingham, Wash., ALR
*Howell, Donald MacLean('31).Polasky Bdlg.,Alma, Mich., ALR
*Hubbard, Wm. B. ('31).....Dryden Bldg., Flint, Mich., ALR
Huber, Walter A.....Medical Arts Bldg., Tulsa, Okla., Op.
Hughes, J. A.('27).....Sterick Bldg., Memphis, Tenn., OALR
*Hughes, L. W.('25).....965 Broad St., Newark, N. J., OALR
Hughes, Ray Whitbeck('28)
1501 David Whitney Bldg., Detroit, Mich., OALR
Hughes, T. E.('22).....Professional Bldg., Richmond, Va., ALR
*Hughes, W. F...401 Hume-Mansur Bldg., Indianapolis, Ind., Op.
*Hughes, Wendell L.('29)
Professional Bldg., Hempstead, L. I., N. Y., Op.
*Huizinga, J. G.('31).Peoples State Bk. Bldg., Holland, Mich., OALR

HULKA, J. H.('34)..30-28 36th St., Long Island City, N. Y., OALR
*HULL, ALLEN('35).....1847 Benson, Ave., Brooklyn, N. Y., Op.
*HUME, JOHN R. ('31)
Maison Blanche Bldg., New Orleans, La., ALR
HUNT, WESTLEY M.('29)...33 E. 68th St., New York, N. Y., ALR
*HUNT, WM. T., JR.('34).....514 Penn St., Huntingdon, Pa., ALR
HUNTER, J. F.('30).....503 Penn Ave., Turtle Creek, Pa., ALR
HUNTER, R. J.('30)....2011 Chestnut St., Philadelphia, Pa., ALR
HURFF, J. WALLACE('19)..86 Washington St., Newark, N. J., OALR
*HURLEY, E. D.('14).....419 Boylston St., Boston, Mass., Op.
*HURST, V. REEVES('22)....Hurst Hospital, Longview, Tex., OALR
HUSIK, D. N.('21).....1930 Chestnut St., Philadelphia, Pa., ALR
HUTCHINSON, ABBOTT TRASK('28)
30 W. 59th St., New York, N. Y., ALR
*HUTCHISON, K. O.('30)
1509 Sherbrooke St., W., Montreal, Can., ALR

I

*IGLAUER, SAMUEL .....707 Race St., Cincinnati, O., ALR
IMPERATORI, C. J. .....108 E. 38th St., New York, N. Y., ALR
INGLIS, HARRY J. .....43 Bay State Road, Boston, Mass., ALR
INGRAM, L. C.('15).....108 E. Central Ave., Orlando, Fla., OALR
IRVIN, EDGAR H. ....903 First Nat'l Bk. Bldg., El Paso, Tex., OALR
IRVINE, A. RAY('18).1144 Roosevelt Bldg., Los Angeles, Calif., Op.
IRVINE, ROBERT S.('29)...490 Post St., San Francisco, Calif., OALR
IRWIN, VINCENT J., JR.('21).1634 Main St., Springfield, Mass., OALR
ISRAEL, SIDNEY....2010 Niels Esperson Bldg., Houston, Tex., ALR
IVY, ROBERT H.('34)....1930 Chestnut St., Philadelphia, Pa., ALR

J

JACKLE, ARTHUR F.('19)..63 E. Fourth St., Dunkirk, N. Y., Op.
*JACKSON, ATRAS EARL('28).Med. Arts Bldg., Ft. Worth, Tex., Op.
*JACKSON, CHEVALIER L. ('34)
3432 N. Broad St., Philadelphia, Pa., L
JACKSON, J. D.('20).....232 West 8th St., Erie, Pa., OALR
JACOBS, MAX W.('16).Missouri Theatre Bldg., St. Louis, Mo., Op.
JACOBS, MILTON('33)...122 S. Michigan Blvd., Chicago, Ill., Op.
JACOBS, S. M.('13).1749 Grand Concourse, New York, N. Y., OALR
JACOBY, DOUGLAS P. A.('12)..106 Touro St., Newport, R. I., Op.AL
*JACOBY, MARK W.('33)...1364 Hanna Bldg., Cleveland, O., Op.
JAMES, WM. M.('31).....508 N. Grand Ave., St. Louis, Mo., Op.
JAMESON, P. CHALMERS('31)
139 Montague St., Brooklyn, N. Y., Op.
*JAQUISH, CHAS. JOS.('28).211 E. Wallace Ave., Newcastle, Pa., ALR
JARVIS, DEFOREST C.('29)....Quarry Bank Bldg., Barre, Vt., ALR
*JENNINGS, CHAS. W.('31)
Hiland Bldg., East End, Pittsburgh, Pa., Op.
JEROME, WM. Z('31)
436 Fort Washington Ave., New York, N. Y., ALR

JERVEY, JAS. W.('35) .....101 Church St., Greenville, S. C., OALR
JESBERG, SIMON('32) .500 S. Lucas Ave., Los Angeles, Calif., ALR
JOHNS, JUANITA PEARL ('33)
313 Commonwealth Ave., Boston, Mass., Op.
JOHNSON, A. EINAR('19)
Kemp and Broadway, Watertown, S. D., OALR
JOHNSON, DAVID M.('32) .....327 E. State St., Columbus, O., ALR
JOHNSON, HENRIETTA MAY('32)
331 S. Lake Ave., Pasadena, Calif., Op.
JOHNSON, HENRY P.('35) .....32 Deering St., Portland, Me., ALR
JOHNSON, H. R.('13) ...320 Jefferson St., Fairmont, W. Va., OALR
JOHNSON, JOHN L.('35) .....33 Grove St., Mangor, Me., ALR
JOHNSON, OTIS H. .....Morehead City, N. C., OALR
JOHNSON, THOMAS H.('23) .30 W. 59th St., New York, N. Y., Op.
*JOHNSTON, KENNETH B.('32)
1509 Sherbrooke St., W., Montreal, Can., Op.
JOHNSTON, W. H.('12) .1515 State St., Santa Barbara, Calif., OALR
JOHNSTON, WILSON ('18)
1927 N. W. Lovejoy St., Portland, Ore., OALR
*JONES, C. C. ('25) .....Doctors Bldg., Cincinnati, O., ALR
*JONES, CECIL CAIN('28) ...Equitable Bldg., Des Moines, Ia., OALR
JONES, DAVID H.('35) .....140 E. 54th St., New York, N. Y., LR
JONES, EDLEY H.('28) .1301 Washington St., Vicksburg, Miss., OALR
JONES, EDMUND L.('26) .Wheeling Clinic, Wheeling, W. Va., OALR
JONES, E. L.('13) ..First Nat'l Bank Bldg., Cumberland, Md., OALR
JONES, E. W. .....44 Atlanta St., Lynn, Mass., Op.A
*JONES, F. W. ('19) .....101 Broadway, Alton, Ill., OALR
JONES, L. W.('25) .....16 N. Goodman St., Rochester, N. Y., OALR
*JONES, M. F.('30) .....121 E. 60th St., New York, N. Y., ALR
*JONES, U. G. ('27)
Cor. Market and Boone Sts., Johnson City, Tenn., ALR
*JONES, VINCENT L. ('28)
1242 Missouri Theatre Bldg., St. Louis, Mo., OALR
*JONES, WM. S. ('27) ..522 Sheridan Rd., Menominee, Mich., OALR
JORDAN, GEO. T.('21) ....30 N. Michigan Blvd., Chicago, Ill., OALR
*JOSEPHSON, EMANUEL M.('29) .108 E. 31st St., New York, N. Y., ALR
JOY, HAROLD H.('26) .504 State Tower Bldg., Syracuse, N. Y., Op.
JOYNT, A. J.('24) .....Black Bldg., Waterloo, Ia., OALR
JOYNT, M. J.('30) .....Royal Bldg., Le Mars, Ia., ALR
*JUDD, DELBERT K.('35) ...Medical Arts Bldg., Omaha, Nebr., ALR
JUDD, H. B.('17) .....40 E. 61st St., New York, N. Y., ALR
*JUDD, J. H.('30) .....Medical Arts Bldg., Omaha, Nebr., Op.
JUDGE, HARRY V.('20) .....46 Willett St., Albany, N. Y., Op.

K

KAHN, M. E.('24) .....104 S. Michigan Ave., Chicago, Ill., ALR
KAMMERLING, THEODORE S.('32)
616 North Central Ave., Chicago, Ill., Op.
KAPLAN, SAMUEL('35) .....1 Nevins St., Brooklyn, N. Y., ALR
KAPP, CARL G.('35) ..440 Westminster Ave., Elizabeth, N. J., ALR

KARKEET, ROBT. B.('28) . Medical Dental Bldg., Portland, Ore., OALR
*KAUFFMAN, A. B.('22) . . 1000 Chimes Bldg., Syracuse, N. Y., ALR
KAUFMAN, A. SPENCER('17)
1923 Spruce St., Philadelphia, Pa., ALR
KAZANJIAN, V. H. ('33)
475 Commonwealth Ave., Boston, Mass., LR
*KEARBY, D. O.('17) . Hume-Mansur Bldg., Indianapolis, Ind., ALR
KEARNY, R. A.('32)
2311 Connecticut Ave., N.W., Washington, D. C., ALR
KEATING, T. F.('27) . . . 1042 Maccabee Bldg., Detroit, Mich., OALR
KEEFE, WM. J.('29) . . 110 E. Sidney Ave., Mt. Vernon, N. Y., ALR
KEELER, LANSING H.('34) . . . 89-04 148th St., Jamaica, N. Y., Op.
KEIM, W. F.('24) . . . . . 25 Roseville Ave., New York, N. Y., ALR
KEIPER, G. F., JR.('30) . . . 727 W. 7th St., Los Angeles, Calif., ALR
KEITH, W. E. . . . . 1000 Professional Bldg., Kansas City, Mo., OALR
KELLER, S. A.('20) . 303 S. Minnesota Ave., Sioux Falls, S. D., OALR
KELLEY, ISAAC D., JR.('28)
3720 Washington Blvd., St. Louis, Mo., ALR
KELLEY, VINCENT J. ('33)
520 Commonwealth Ave., Boston, Mass., ALR
KELLY, GEO. F. J. ('34)
Suite 700, N.E. Cor. 20th & Chestnut Sts., Philadelphia, Pa., Op.
KELLY, JOSEPH D.('28) . . . 140 E. 54th St., New York, N. Y., OALR
KEMLER, JOS. I.('22) . . . . . 1908 Eutaw Pl., Baltimore, Md., OALR
KEND, LEE('32) . . . . . 405 Park Ave., New York, N. Y., ALR
KENDALL, M. RAYMOND('19) . 2474 S. Taylor Rd.,Cleveland,O., ALR
KENNEDY, ELDRED WESTON('28)
707 Gas and Electric Bldg., Rochester, N. Y., Op.
*KENNON, W. G.('24) . . . . . Doctors' Bldg., Nashville, Tenn., OALR
*KERSHNER, W. E. ('34) . . . . . 117 Front St., Bath, Me., ALR
KETTELKAMP, FRED O.('26)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
KEY, BEN WITT('14) . . . . . 100 W. 59th St., New York, N. Y., Op.
KEY, SAM N.('26) . . . . . Norwood Bldg., Austin, Tex., OALR
*KEYES, JOHN E. L.('33) . Home S.&L. Bldg., Youngstown, O., Op.
*KEYS, MORTON J.('22) . Pemberton Bldg., Victoria, B. C., Can., OALR
KEYTON, JOHN A.('34) . . . . . 108½ E. Main St., Dothan, Ala., ALR
KIEFER, HUGO A.('18) . . Brockman Bldg., Los Angeles, Calif., OALR
KIEHLE, F. A.('15) . . . 1020 S. W. Taylor St., Portland, Ore., OALR
*KILBOURNE, P. H. ('24) . . . . Fidelity Med. Bldg., Dayton, O., OALR
*KILGORE, GEO. L.('35) . Bk. of Amer. Bldg., San Diego, Calif., Op.
*KILLEEN, JOHN JOS.('28) . 104 S. Michigan Ave., Chicago, Ill., ALR
KINCANNON, LEROY TAYLOR('28)
1927 First Ave. N., Birmingham, Ala., OALR
KING, CHAS. R.('20) . . . . . Spitzer Bldg., Toledo, O., OALR
*KING, ED. . . . . 1002 Carew Tower, Cincinnati, O., ALR
*KING, GEO. L., JR.('29) . . . . . 537 E. Market St., Alliance, O., Op.
KING, JAMES J. ('16) . . . . . 133 E. 58th St., New York, N. Y., ALR
*KING, LOWELL W.('33) . . . . . 470 E. State St., Salem, O., ALR
KING, O. H. ('16) . . . . Medical Arts Bldg., Hot Springs, Ark., OALR

*KINNE, SANDFORD H.('28) . .73 Main St., Binghamton, N. Y., ALR
KINNEY, CHAS. ED.('34) .10515 Carnegie Ave., Cleveland, O., ALR
*KIRBY, D. B.('24) . . . . . .780 Park Ave., New York, N. Y., Op.
KIRKBRIGHT, JAS. C.('35) . . .40 E. 61st St., New York, N. Y., ALR
KISTLER, O. F. . . . . .43 N. Franklin St., Wilkes-Barre, Pa., Op.AR
KISTNER, F. B. . . . . .600 Mayer Bldg., Portland, Ore., OALR
KITTREDGE, FRANK E.('31) .Masonic Temple, Nashua, N. H., ALR
KLEINERT, MARGARET NOYES
82 Commonwealth Ave., Boston, Mass., ALR
KLINE, H. G.('12) . . . . . .State Tower Bldg., Syracuse, N. Y., ALR
KLINE, O. R.('30) . . . . . .414 Cooper St., Camden, N. J., ALR
KLINGER, THOMAS O.('20) .Med. Arts Bldg., Springfield, Mo., OALR
*KNAPP, ARTHUR A.('33) . . .133 E. 58th St., New York, N. Y., Op.
KNAPP, B. J. . . . . .Citizens' Bank Bldg., Evansville, Ind., OALR
KNAPP, F. N.('25) . . . . . .Medical Arts Bldg., Duluth, Minn., OALR
*KNAUER, WM. J.('30) . . . .Buckman Bldg., Jacksonville, Fla., OALR
*KNIGHT, JOHN S.('32) .Professional Bldg., Kansas City, Mo., ALR
KNIGHTON, WILLIS S.('31) .40 E. 61st St., New York, N. Y., Op.
KNODE, A. R.('16) . . . .708 Medical Arts Bldg., Omaha, Nebr., ALR
KNOWLTON, CHAS. D. ('29)
520 Commonwealth Ave., Boston, Mass., ALR
*KOEBBE, EZRA E.('29) .Columbus Bk. Bldg., Columbus, Nebr., ALR
*KOENIG, IVAN J.('28) . . . . . .40 North St., Buffalo, N. Y., Op.
*KOLLER, LOUIS R.('32) .Med. Arts Bldg., Minneapolis, Minn., ALR
KOOBS, HERMAN J. G.('20) .117½ W. Walnut St., Rogers,Ark., OALR
KRAFT, OSCAR H.('12) .1518 N. Dearborn Pkwy., Chicago, Ill., Op.
*KRAMER, JULIUS C.('34) . . .130 E. Market St., Sandusky, O., ALR
KRAUSS, FREDERICK. . . .1930 Chestnut St., Philadelphia, Pa., OALR
KREBS, ADOLPH('13) . . . . . .Jenkins Arcade, Pittsburgh, Pa., Op.
KRESS, GEO. H.('18) . . . .Roosevelt Bldg., Los Angeles, Calif., OALR
*KREUTZ, G. C.('30) . . . .Henry Ford Hospital, Detroit, Mich., ALR
KRIEGER, WM. A.('19) .103 Hooker Ave., Poughkeepsie, N. Y., OALR
KRIMSKY, EMANUEL('32) .745 Eastern Pkwy, Brooklyn, N.Y., Op.
KRIMSKY, JOSEPH('28) . . .302 Empire Blvd., Brooklyn, N.Y., OALR
KRONFELD, PETER C. ('31)
Peiping Union Medical College, Peiping, China, Op.
KRUSKAL, I. D.('25) . . .101 Lafayette Ave., Brooklyn, N. Y., Op.
KUHN, H. S.('28) . . . . .5231 Hohman Ave., Hammond, Ind., Op.
*KUHN, HUGH A.('28) . .5231 Hohman Ave., Hammond, Ind., ALR
KULLY, B. M.('22) . .3875 Wilshire Blvd., Los Angeles, Calif., ALR
*KULLY, HERMAN ED.('35) .Medical Arts Bldg., Omaha, Nebr., ALR
KULVIN, MAX M.('31) . . .55 E. Washington St., Chicago, Ill., OALR

L

LAESSLE, HENRY A.('22) .5900 Market St., Philadelphia, Pa., ALR
*LAFF, H. I.('30) . . . . . .Metropolitan Bldg., Denver, Colo., ALR
LAIRD, ROBERT G.('32) . . . .Metz Bldg., Grand Rapids, Mich., Op.
LAMB, HARVEY D.('20) . . .Metropolitan Bldg., St. Louis, Mo., Op.
*LAMB, HARRY H.('29) . .Davenport Bk. Bldg., Davenport, Ia., ALR
LAMBERT, ROBERT K.('32) .1010 Fifth Ave., New York, N.Y., Op.

KARKEET, ROBT. B.('28) . Medical Dental Bldg., Portland, Ore., OALR
*KAUFFMAN, A. B.('22) . . 1000 Chimes Bldg., Syracuse, N. Y., ALR
KAUFMAN, A. SPENCER('17)
1923 Spruce St., Philadelphia, Pa., ALR
KAZANJIAN, V. H. ('33)
475 Commonwealth Ave., Boston, Mass., LR
*KEARBY, D. O.('17) . Hume-Mansur Bldg., Indianapolis, Ind., ALR
KEARNY, R. A.('32)
2311 Connecticut Ave., N.W., Washington, D. C., ALR
KEATING, T. F.('27) . . . 1042 Maccabee Bldg., Detroit, Mich., OALR
KEEFE, WM. J.('29) . . 110 E. Sidney Ave., Mt. Vernon, N. Y., ALR
KEELER, LANSING H.('34) . . . 89-04 148th St., Jamaica, N. Y., Op.
KEIM, W. F.('24) . . . . . 25 Roseville Ave., New York, N. Y., ALR
KEIPER, G. F., JR.('30) . . . 727 W. 7th St., Los Angeles, Calif., ALR
KEITH, W. E. . . . . 1000 Professional Bldg., Kansas City, Mo., OALR
KELLER, S. A.('20) . 303 S. Minnesota Ave., Sioux Falls, S. D., OALR
KELLEY, ISAAC D., JR.('28)
3720 Washington Blvd., St. Louis, Mo., ALR
KELLEY, VINCENT J. ('33)
520 Commonwealth Ave., Boston, Mass., ALR
KELLY, GEO. F. J. ('34)
Suite 700, N.E. Cor. 20th & Chestnut Sts., Philadelphia, Pa., Op.
KELLY, JOSEPH D.('28) . . . 140 E. 54th St., New York, N. Y., OALR
KEMLER, JOS. I.('22) . . . . . 1908 Eutaw Pl., Baltimore, Md., OALR
KEND, LEE('32) . . . . . 405 Park Ave., New York, N. Y., ALR
KENDALL, M. RAYMOND('19) . 2474 S. Taylor Rd., Cleveland, O., ALR
KENNEDY, ELDRED WESTON('28)
707 Gas and Electric Bldg., Rochester, N. Y., Op.
*KENNON, W. G.('24) . . . . . Doctors' Bldg., Nashville, Tenn., OALR
*KERSHNER, W. E. ('34) . . . . . 117 Front St., Bath, Me., ALR
KETTELKAMP, FRED O.('26)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
KEY, BEN WITT('14) . . . . . 100 W. 59th St., New York, N. Y., Op.
KEY, SAM N.('26) . . . . . Norwood Bldg., Austin, Tex., OALR
*KEYES, JOHN E. L.('33) . Home S.&L. Bldg., Youngstown, O., Op.
*KEYS, MORTON J.('22) . Pemberton Bldg., Victoria, B. C., Can., OALR
KEYTON, JOHN A.('34) . . . . . 108½ E. Main St., Dothan, Ala., ALR
KIEFER, HUGO A.('18) . . Brockman Bldg., Los Angeles, Calif., OALR
KIEHLE, F. A.('15) . . . 1020 S. W. Taylor St., Portland, Ore., OALR
*KILBOURNE, P. H. ('24) . . . . Fidelity Med. Bldg., Dayton, O., OALR
*KILGORE, GEO. L.('35) . Bk. of Amer. Bldg., San Diego, Calif., Op.
*KILLEEN, JOHN JOS.('28) . 104 S. Michigan Ave., Chicago, Ill., ALR
KINCANNON, LEROY TAYLOR('28)
1927 First Ave. N., Birmingham, Ala., OALR
KING, CHAS. R.('20) . . . . . Spitzer Bldg., Toledo, O., OALR
*KING, ED. . . . . 1002 Carew Tower, Cincinnati, O., ALR
*KING, GEO. L., JR.('29) . . . . . 537 E. Market St., Alliance, O., Op.
KING, JAMES J. ('16) . . . . . 133 E. 58th St., New York, N. Y., ALR
*KING, LOWELL W.('33) . . . . . 470 E. State St., Salem, O., ALR
KING, O. H. ('16) . . . . Medical Arts Bldg., Hot Springs, Ark., OALR

*KINNE, SANDFORD H.('28) . . 73 Main St., Binghamton, N. Y., ALR
KINNEY, CHAS. ED.('34) . 10515 Carnegie Ave., Cleveland, O., ALR
*KIRBY, D. B.('24) . . . . . 780 Park Ave., New York, N. Y., Op.
KIRKBRIGHT, JAS. C.('35) . . . 40 E. 61st St., New York, N. Y., ALR
KISTLER, O. F. . . . . 43 N. Franklin St., Wilkes-Barre, Pa., Op.AR
KISTNER, F. B. . . . . 600 Mayer Bldg., Portland, Ore., OALR
KITTREDGE, FRANK E.('31) . Masonic Temple, Nashua, N. H., ALR
KLEINERT, MARGARET NOYES
82 Commonwealth Ave., Boston, Mass., ALR
KLINE, H. G.('12) . . . . . State Tower Bldg., Syracuse, N. Y., ALR
KLINE, O. R.('30) . . . . . 414 Cooper St., Camden, N. J., ALR
KLINGER, THOMAS O.('20) . Med. Arts Bldg., Springfield, Mo., OALR
*KNAPP, ARTHUR A.('33) . . . 133 E. 58th St., New York, N. Y., Op.
KNAPP, B. J. . . . . Citizens' Bank Bldg., Evansville, Ind., OALR
KNAPP, F. N.('25) . . . . . Medical Arts Bldg., Duluth, Minn., OALR
*KNAUER, WM. J.('30) . . . . Buckman Bldg., Jacksonville, Fla., OALR
*KNIGHT, JOHN S.('32) . Professional Bldg., Kansas City, Mo., ALR
KNIGHTON, WILLIS S.('31) . 40 E. 61st St., New York, N. Y., Op.
KNODE, A. R.('16) . . . . 708 Medical Arts Bldg., Omaha, Nebr., ALR
KNOWLTON, CHAS. D. ('29)
520 Commonwealth Ave., Boston, Mass., ALR
*KOEBBE, EZRA E.('29) . Columbus Bk. Bldg., Columbus, Nebr., ALR
*KOENIG, IVAN J.('28) . . . . . 40 North St., Buffalo, N. Y., Op.
*KOLLER, LOUIS R.('32) . Med. Arts Bldg., Minneapolis, Minn., ALR
KOOBS, HERMAN J. G.('20) . 117½ W. Walnut St., Rogers,Ark., OALR
KRAFT, OSCAR H.('12) . 1518 N. Dearborn Pkwy., Chicago, Ill., Op.
*KRAMER, JULIUS C.('34) . . . 130 E. Market St., Sandusky, O., ALR
KRAUSS, FREDERICK. . . . 1930 Chestnut St., Philadelphia, Pa., OALR
KREBS, ADOLPH('13) . . . . . Jenkins Arcade, Pittsburgh, Pa., Op.
KRESS, GEO. H.('18) . . . . Roosevelt Bldg., Los Angeles, Calif., OALR
*KREUTZ, G. C.('30) . . . . Henry Ford Hospital, Detroit, Mich., ALR
KRIEGER, WM. A.('19) . 103 Hooker Ave., Poughkeepsie, N. Y., OALR
KRIMSKY, EMANUEL('32) . 745 Eastern Pkwy, Brooklyn, N.Y., Op.
KRIMSKY, JOSEPH('28) . . . 302 Empire Blvd., Brooklyn, N.Y., OALR
KRONFELD, PETER C. ('31)
Peiping Union Medical College, Peiping, China, Op.
KRUSKAL, I. D.('25) . . . 101 Lafayette Ave., Brooklyn, N. Y., Op.
KUHN, H. S.('28) . . . . . 5231 Hohman Ave., Hammond, Ind., Op.
*KUHN, HUGH A.('28) . . 5231 Hohman Ave., Hammond, Ind., ALR
KULLY, B. M.('22) . . 3875 Wilshire Blvd., Los Angeles, Calif., ALR
*KULLY, HERMAN ED.('35) . Medical Arts Bldg., Omaha, Nebr., ALR
KULVIN, MAX M.('31) . . . 55 E. Washington St., Chicago, Ill., OALR

L

LAESSLE, HENRY A.('22) . 5900 Market St., Philadelphia, Pa., ALR
*LAFF, H. I.('30) . . . . . Metropolitan Bldg., Denver, Colo., ALR
LAIRD, ROBERT G.('32) . . . . Metz Bldg., Grand Rapids, Mich., Op.
LAMB, HARVEY D.('20) . . . Metropolitan Bldg., St. Louis, Mo., Op.
*LAMB, HARRY H.('29) . . Davenport Bk. Bldg., Davenport, Ia., ALR
LAMBERT, ROBERT K.('32) . 1010 Fifth Ave., New York, N.Y., Op.

LaMotte, Wm. O.('35)..Med. Arts Bldg., Wilmington, Del., ALR
Lampert, Max T.('31).....Northwest Clinic, Minot, N. D., ALR
Lancaster, W. B.('13).520 Commonwealth Ave., Boston, Mass., Op.
Landfried, C. J. .....607 Baronne Bldg., New Orleans, La., ALR
*Landis, J. E. ('30).....517 Walnut St., Reading, Pa., ALR
Lane, Franklin F.('33), U. S. S. Minneapolis
care Postmaster, San Pedro, Calif., OALR
*Lang, Geo. Hermanne('32).202 E. Liberty St.,Savannah,Ga., ALR
Laning, J. Halcombe('18).907 Rialto Bldg., KansasCity,Mo., ALR
LaReau, Hector G.('35)...4803 Lincoln Ave., Chicago, Ill., ALR
*Larkin, Bernard J.('16)..23 E. Ohio St., Indianapolis, Ind., Op.
Larsen, Carl L.('16).1027 Lowry Med. Bldg.,St Paul,Minn., OALR
*Larson, G. Arthur('33)....A.O.U.W. Bldg., Fargo, N. D., Op.
*LaRue, C. L.('22).....Highland Clinic, Shreveport, La., OALR
Lassen, Fritz(15).....Pueblo Clinic Bldg., Pueblo, Colo., OALR
La Vigne, A. A.....471 Park Ave., New York, N. Y., OALR
Lawrence, Granville A.('13)
Medical Arts Bldg., Philadelphia, Pa., Op.
*Lawson, Lawrence J.('31)...636 Church St., Evanston, Ill., ALR
*Lawwill, Stewart('28).Med. Arts Bldg., Chattanooga,Tenn., OALR
*Leasure, John K.('31).Hume-Mansur Bldg., Indianapolis,Ind., ALR
Leavenworth, R. O.('30)
Lowry Medical Arts Bldg., St. Paul, Minn., Op.
Leavy, C. A. .....Metropolitan Bldg., St. Louis, Mo., ALR
*Lebensohn, Jas. E.('27).310 S. Michigan Ave., Chicago, Ill., OALR
Le Compte, E. D.('27).9 Exchange Pl., Salt Lake City, Utah, OALR
*Lederer, Francis L.('25).307 N. Michigan Blvd.,Chicago,Ill., ALR
Leech, J. W.('14).....167 Angell St., Providence, R. I., OALR
*Lefevre, Willis H.('35)...234 N. Duke St., Lancaster, Pa., ALR
*Leech, V. M. ('30).....55 E. Washington St., Chicago, Ill., Op.
Lefler, Anna B.('18).610 S. Broadway, Los Angeles, Calif., OALR
*Leggett, Albert E.('34)..614 Breslin Bldg., Louisville, Ky., Op.
Lehan, James W.('20)....Park Place Bldg., Greeley, Colo., OALR
Leibowitz, Philip('33)...135 E. Parkway, Brooklyn, N. Y., ALR
LeJeune, F. E.('27)
632 Maison Blanche Bldg., New Orleans, La., ALR
Lemel, Samuel L.('35)....1148 Euclid Ave., Cleveland, O., ALR
Lemere, H. B.('12).9615 Brighton Way, Beverly Hills, Calif., OALR
Lemoine, A. N.('20).....Rialto Bldg., Kansas City, Mo., Op.
Lent, E. J.....122 N. Lafayette Blvd., South Bend, Ind., OALR
Lerner, Macy Levi('23)..332 Park Ave., Rochester, N. Y., Op.
*Leshin, Norman('31)..180 N. Michigan Ave., Chicago, Ill., ALR
Levine, Joseph('31).....105 E. 63rd St., New York, N. Y., Op.
Levitt, Jesse M.('35).....734 Ocean Ave., Brooklyn, N. Y., Op.
*Levy, Abram('34).....507 Church St., Bound Brook, N. J., Op.
Levy, Harry H. .....State Tower Bldg., Syracuse, N. Y., Op.
*Levy, Louis('16)...Commerce Title Bldg., Memphis, Tenn., ALR
*Le Win, Thurber('29)...112 Linwood Ave., Buffalo, N. Y., Op.
*Lewis, A. C. ('16).....130 Madison Ave., Memphis, Tenn., OALR
Lewis, Fielding O. .....259 S. 17th St., Philadelphia, Pa., ALR

LEWIS, GEORGE A.('20).....213 Main St., Roundup, Mont., OALR
LEWIS, J. D.('21)....1534 Estado St., Santa Barbara, Calif., OALR
LEWIS, J. P.('15)...479 Commonwealth Ave., Boston, Mass., ALR
and 681 Main St., Waltham, Mass.
LEWIS, PHILIP M('28)....Exchange Bldg., Memphis, Tenn., OALR
LEYDA, JAMES H.('27)....Metropolitan Bldg., Denver, Colo., ALR
LIDIKAY, C. J. ('20).....903 N. 7th St., Kansas City, Kan., OALR
*LIERLE, DEAN M.('28)..University Hospital, Iowa City, Ia., ALR
LIFSCHUTZ, JACOB('25)..310 S. Michigan Ave., Chicago, Ill., OALR
*LILLIE, HAROLD I. .....Mayo Clinic, Mochester, Minn., ALR
LILLIE, W. IVAN('30)..3701 N. Broad St., Philadelphia, Pa., Op.
*LINGEMAN, B. N.('30)..Ben Hur Bldg., Crawfordsville, Ind., ALR
*LINGEMAN, ED. L.('29).406 Pennway Bldg.,Indianapolis,Ind., ALR
*LINN, J. G.('19).....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*LISCHKOFF, M. A.('13).....Blount Bldg., Pensacola, Fla., OALR
LITCHFIELD, J. T.('21).Med. Arts Bldg., Minneapolis, Minn., OALR
*LITTELL, JOS. J.('31).Hume-Mansur Bldg., Indianapolis, Ind., ALR
LITTWIN, CHAS.('33).33 E. Palisade Ave., Englewood, N. J., Op.
also 550 Park Ave., New York, N. Y.
*LIVINGSTON, GEO. S.('30).185 N. Wabash Ave., Chicago, Ill., ALR
LLOYD, A. W. ('15).....804 Lloyd Bldg., Hammond, Ind., OALR
LLOYD, RALPH I.('25).....14 Eighth Ave., Brooklyn, N. Y., Op.
LOEWE,WALTER R.('34).1114 Madison Ave.,New York,N.Y., Op.
LOFTUS, JOHN ED.('22).1930 Chestnut St., Philadelphia, Pa., ALR
LOGUE, LYLE J.('31).....1304 Walker St., Houston, Texas, ALR
LOKEY, H. M. .....Medical Arts Bldg., Atlanta, Ga., OALR
LOMBARDO, MELCHIORE('23).142 Joralemon St., Brooklyn,N.Y., Op.
LOOMIS, E. A.('18)..Medical Arts Bldg., Minneapolis, Minn., OALR
LOOPER, EDWARD A.('23).104 W. Madison St., Baltimore, Md., OALR
LORIE, ALVIN J.('17)....Commerce Bldg., Kansas City, Mo., ALR
LORING, J. BROWN('08)..25 E. Washington St., Chicago, Ill., OALR
LOVE, JOHN G.('20).....Ilgenfritz Bldg., Sedalia, Mo., OALR
LOWELL, W. HOLBROOK('31)
82 Commonwealth Ave., Boston, Mass., Op.
*LOWENTHAL, GERSON('35)..19 Garfield Place, Cincinnati, O., ALR
LOWRY, B. W.('25).....205 Zack St., Tampa, Fla., OALR
LUBMAN, MAX('19).....118 E. 93rd St., New York, N. Y., ALR
LUCAS, HAROLD ROY('28).Med.-Dental Bldg., Portland, Ore., OALR
LUEDDE, W. H.('16).....Metropolitan Bldg., St. Louis, Mo., Op.
*LUHR, ALFRED F. ('31)....40 W. North St., Buffalo, N. Y., Op.
*LUKENS, J. A. ('30).....316 Michigan St., Toledo, O., ALR
LUKENS, ROBERT McD. ...1923 Spruce St., Philadelphia, Pa., ALR
LUNDON, A. E.('27)...1501 Crescent St., Montreal, Canada, ALR
LUONGO, ROMEO A.('31)....1713 Pine St., Philadelphia, Pa., ALR
*LUPO, CARL W.('32).....7425 Ridge Blvd., Brooklyn, N. Y., ALR
LUPTON, IRVING M.('28).806 Med. Arts Bldg.,Portland,Ore., OALR
*LURIE, M. H.('30).....483 Beacon St., Boston, Mass., ALR
LUX, PAUL ('25).....2006 Bryant Bldg., Kansas City, Mo., ALR
*LYLE, DONALD J. ('27).....Carew Tower, Cincinnati, O., Op.
*LYMAN, HARRY W. '(23).....308 N. 6th St., St. Louis, Mo., ALR

*LYNCH, ROBERT J.('31) . . . .Equitable Bldg., Des Moines, Ia., ALR
LYONS, H. R. . . . . .30 N. Michigan Blvd., Chicago, Ill., ALR

Mc

MCADAMS, WM. R.('33) . . . .704 Congress St., Portland, Me., Op.
MCALESTER, ANDREW W., JR. .Bryant Bldg., Kansas City, Mo., Op.
*MCALESTER, ANDREW W., 3RD ('32)
Bryant Bldg., Kansas City, Mo., Op.
MCANDREWS, LEO F.('31) .1930 Chestnut St.,Philadelphia,Pa., Op.
MCASKILL, JAS E.('32) .Woolworth Bldg., Watertown, N. Y., ALR
MCAULIFFE, G. W.('32) . .110 W. 55th St., New York, N. Y., ALR
MCBEAN, GEO. M. . . . . .25 E. Washington St., Chicago, Ill., OALR
MCBRIDE, W. O.('17) .2902 S. Fairfield Ave., Ft. Wayne, Ind., OALR
*MCCANNEL, A. D. . . . . .15A Central Ave. W., Minot, N. D., OALR
MCCARTNEY, OLIVER P.('20) .Valley Bank Bldg., Mesa, Ariz., OALR
MCCARTY, VIRGIL W.('20) .Professional Bldg.,Kansas City,Mo., OALR
*MCCAW, JOHN A. . . . . .418 Majestic Bldg., Denver, Colo., Op.
MCCLELLAN, W. E. ('27) . . . .750 Main St., Hartford, Conn., OALR
MCCLELLAND, CARL C.('23)
1857 David Whitney Bldg., Detroit, Mich., OALR
*MCCOLLOUGH, T. B.('32) .121 University Pl., Pittsburgh, Pa., ALR
MCCORMICK, T. F.('30) . . . . . .Wells Bldg., Milwaukee, Wis., ALR
MCCOY, L. L.('30) . . . . . .817 Summit Ave., Seattle, Wash., Op.
*MCCREADY, J. HOMER. . . .121 University Pl., Pittsburgh, Pa., ALR
*MCCULLOUGH, ROBERT J. P.('20)
830 Medical Arts Bldg., Toronto, Can., OALR
*MCCULLOUGH, C. J. ('29)
Washington Trust Bldg., Washington, Pa., ALR
MCDANNALD, C. E.('27) . .100 W. 59th St., New York, N.Y., Op.
MCDAVITT, BERTHA SCHENCK STECKER('29)
Main and Avenue A. Temple, Texas, ALR
MCDERMOTT, JOHN J.('23) .1821 Niles Ave., St. Joseph, Mich., OALR
MCDIARMID, H. OLIVER('18)
35 Clement Block, Brandon, Manitoba, Can., OALR
MCDONALD, C. L. . . . . .964 Rose Bldg., Cleveland, O., OALR
MCDONALD, DENNIS J.('26) .17 E. 38th St., New York, N. Y., ALR
MCDOUGALL, J. CALHOUN('32) .Med. Arts Bldg., Atlanta, Ga., ALR
MCDOUGALL, WM. L.('31) . . . . . .Doctors Bldg., Atlanta, Ga., ALR
MCDOWELL, N. D.('14) .275 Alexander St., Rochester, N. Y., OALR
*MCGARVEY, WM. ED.('29)
Jackson City Bank Bldg., Jackson, Mich., ALR
MCGILLICUDDY, O. B.('30) . .124 W. Allegan, Lansing, Mich., ALR
MCGIVERN, C. S.('22) . .805 Pacific Ave., Atlantic City, N. J., ALR
*MCGREGOR, GREGOR('31) . .170 St. George St., Toronto, Can., ALR
MCGUIRE, H. H.('13) . .105 N. Braddock St., Winchester, Va., OALR
*MCHENRY, L. CHESTER('30)
Medical Arts Bldg., Oklahoma City, Okla., ALR
MCKEE, CLAUD W.('17) . . . . .Coulter Bldg., Greensburg, Pa., OALR
MCKEE, JOS. W.('28) .Professional Bldg., Kansas City, Mo., Op.
MCKEE, OTTO S.('28) . . . . . .83 Bryant St., Buffalo, N. Y., ALR

McKennan, James W.
514 Washington Trust Bldg., Washington, Pa., Op.
McKeown, Elmer E. .....Republic Bldg., Denver, Colo., OALR
McKeown, Hugh S.('31) .30 W. 59th St., New York, N. Y., Op.
also Bronxville, N. Y.,
*McKinney, A. R.('23)
704 Second Nat'l Bank Bldg., Saginaw, Mich., Op.
McKinney, R.('08) ....899 Madison Ave., Memphis, Tenn., ALR
McLaughlin, P. A..71 S. Washington St.,Wilkes-Barre,Pa., OALR
McLaurin, John G.('26)..Medical Arts Bldg., Dallas, Tex., OALR
McLellan, Thomas G.('35)
First Nat'l Bank Bldg., Connellsville, Pa., Op.
McLoone, John J.('26)..Professional Bldg., Phoenix, Ariz., OALR
McMahon, Bernard John('26)
Missouri Theater Bldg., St. Louis., Mo., ALR
*McMurray, J. B..Washington Trust Bldg., Washington, Pa., OALR
*McNally, Wm. James('28)...Linton Apts., Montreal, Can., ALR
McNichols, W. A.('25).....101 W. 1st St., Dixon, Ill., OALR
*McRae, John H.('30).Med. Arts Bldg.,Grand Rapids,Mich., Op.
McWilliams, Chas. A.('27).G.&S.I. Bldg., Gulfport, Miss., ALR
MacCready, Paul B.('31).442 Temple St.,New Haven,Conn., ALR
*Macdonald, J.C.('30).301 N.W.12th St.,Oklahoma City,Okla., ALR
MacDonald, Philip G.('35).115 E. 61st St., New York, N.Y., ALR
Macfarlane, P. B...Med. Arts Bldg., Hamilton, Ont., Can., OALR
Mackenzie, Geo. W.('12).269 S. 19th St., Philadelphia, Pa., OALR
MacLachlan, A. A.....May Bldg., Pittsburgh, Pa., ALR
*MacLean, Angus L.('35)..1201 N. Calvert, Baltimore, Md., Op.
MacMillan, J. A.('25)....1410 Stanley St., Montreal, Can., Op.
Macnaughton, B. F.('32)
Drummond Medical Bldg., Montreal, Can., ALR
Macnie, John P.('35)..635 W. 165th St., New York, N. Y., Op.
Macnie, J. S. .....Medical Arts Bldg., Minneapolis, Minn., OALR

M

Madden, Reed .....Allen Bldg., Xenia, O., OALR
Maggard, D. I.('20).....715 Orpheum Bldg., Wichita, Kan., OALR
Maghy, Chas. A.('26)
Wilshire Medical Bldg., Los Angeles, Calif., Op.
Mahoney, Paul Leo('28).Donaghey Bldg., Little Rock,Ark., ALR
Maiden, Sydner D.('18).403 Bennett Bldg.,Council Bluffs,Ia., OALR
Malone, F. B.('30).....1917 Tenth St., Lubbock, Texas, ALR
Maloy, Wayland H.('33)
608½ W. Sheridan Ave., Shenandoah, Iowa, Op.
*Mann, Albert H.('35)..209 Pine St., Texarkana, Ark.Tex., ALR
Mann, B. H. ('30)
N.E. corner 20th and Chestnut Sts., Philadelphia, Pa., Op.
*Mann, J. J.('34).....255 State St., Perth Amboy, N. J., ALR
*March, Clara A.('22)...465 Ashland Ave., Buffalo, N. Y., Op.
*Marchman, Oscar M.('35).Medical Arts Bldg., Dallas, Tex., ALR
Marcotte, Raymond H.('32).215A Main St., Nashua, N. H., ALR

MARKEL, J. CLYDE ('17) . Westinghouse Bldg., Pittsburgh, Pa., Op.
MARKS, JULIAN B. ('29) . 735 N. Water St., Milwaukee, Wis., ALR
*MARKS, SAM'L B. ('28) . Security Trust Bldg., Lexington, Ky., ALR
*MARLOW, SEARLE B. ('25)
1004 State Tower Bldg., Syracuse, N. Y., Op.
MARSDEN, W. C. ('30) . . 99 N. Franklin St., Wilkes-Barre, Pa., ALR
MARSHALL, GEORGE GUERIN ('23) . Gryphon Bldg., Rutland, Vt., OALR
*MARSHALL, HAL E. ('35) . . 227 E. Douglas St., Wichita, Kan., ALR
*MARSHALL, WATSON . . . . . 121 University Pl., Pittsburgh, Pa., ALR
MARTIN, ROY D. ('29) . . . . . Foote Bldg., Hastings, Nebr., ALR
*MARTIN, W. O., JR. ('30) . . . . . Doctors Bldg., Atlanta, Ga., Op.
MASON, ROY EARL ('28) . . . . . Frisco Bldg., St. Louis, Mo., Op.
*MASTERS, ROBT. JOHN ('28) . 23 E. Ohio St., Indianapolis, Ind., Op.
MATHESON, J. P. ('15) . . . . . 106 West 7th St., Charlotte, N. C., ALR
MATTHEWS, JUSTUS . . Medical Arts Bldg., Minneapolis, Minn., LR
MATUSOW, A. P. ('35) . 1100 Grand Concourse, New York, N. Y., ALR
MAUMENEE, A. F. . . . . 711 Empire Bldg., Birmingham, Ala., OALR
MAXWELL, HARVEY C. ('35) . Sta. Hosp., Ft. Sam Houston, Tex., OALR
MAXWELL, JAS. H. ('34) . 1st Nat'l Bk. Bldg., Ann Arbor, Mich., ALR
MAY, J. V. ('15) . . . . . 1703 Main St., Marinette, Wis., OALR
MAYBAUM, J. L. ('16) . . . . 1070 Park Ave., New York, N. Y., ALR
*MAYER, LEO LOEB ('28) . . 104 S. Michigan Ave., Chicago, Ill., Op.
MAYERBERG, EMIL R. ('29) . Med. Arts Bldg., Wilmington, Del., ALR
MEANOR, WM. C. . . . . 1715 Clark Bldg., Pittsburgh, Pa., Op.
MEANS, R. G. ('30) . . . . . 40 S. Third St., Columbus, O., ALR
MEEK, RAYMOND E. ('31) . . . 729 Park Ave., New York, N. Y., Op.
MELLINGER, WM. J. ('27) . 120 E. Sola St., Santa Barbara, Calif., ALR
MELOY, WM. C. ('33) . . 2019 R. St., N.W., Washington, D. C., ALR
MELTON, BERNARD L., JR. ('31) . 15 E. Monroe, Phoenix, Ariz., Op.
*MELTZER, PHILIP E. ('34) . 20 Charlesgate W., Boston, Mass., ALR
MENDEL, JAS. H. ('32)
Germantown Prof. Bldg., Philadelphia, Pa., ALR
MENGER, LOUIS C. ('29) . . . 202 Weirfield St., Brooklyn, N. Y., ALR
MERCER, RAY ('27) . . . . . W. C. U. Bldg., Quincy, Ill., OALR
*MERICA, F. W. ('30) . . . . . 14805 Detroit Ave., Lakewood, O., ALR
MERKEL, CHARLES CARSON ('28)
279 Rivard Blvd., Grosse Pointe Village, Mich., ALR
MERRILL, H. G. ('18) . . . . 35 East 2nd South St., Provo, Utah, OALR
MERTINS, PAUL S. ('28) . . . 1115 Bell Bldg., Montgomery, Ala., OALR
MESIROW, MAURICE E. ('35) . Rubel Bldg., Santa Maria, Calif., Op.
MESSIER, A. EUGENE ('25) . . 390 Main St., Worcester, Mass., OALR
MESSINGER, H. C. ('14) . . . . 210 Angell St., Providence, R. I., Op.
*MEYER, SAMUEL JAS. ('28) . 58 E. Washington St., Chicago, Ill., Op.
MIDDLETON, A. B. . . . . Sterry Block, Pontiac, Ill., OALR
*MIDDLETON, HARRY E. ('20) . . . . . Times Bldg., Alton, Ill., OALR
MILLER, ALICE R. ('19) . 418 Fern St., West Palm Beach, Fla., OALR
MILLER, CLIFTON M. . . Stuart Circle Hospital, Richmond, Va., OALR
*MILLER, G. LESLIE ('35) . . . 14805 Detroit Ave., Lakewood, O., Op.
MILLER, H. ED. ('30) . . . . . Missouri Bldg., St. Louis, Mo., ALR
MILLER, HUGH ('20) . . . . . Argyle Bldg., Kansas City, Mo., OALR

MILLER, JOHN ('30) .....63 North St., Greenwich, Conn., ALR
MILLER, JOSEPH A. ('32) .364 Prospect St., South Orange, N.J., ALR
MILLER, J. W. ('29) .....305 E. 17th St., New York, N. Y., ALR
MILLER, M. VALENTINE ('34)
114 W. Phil-Ellena St., Mt. Airy, Philadelphia, Pa., ALR
MILLER, SAUL ('34) .....8805 150th St., Jamaica, N. Y., Op.
MILLER, THEO. E. ('31) ...122 S. Michigan Ave., Chicago, Ill., ALR
*MILLER, WM. H. ('31) .....Tribune Bldg., Terre Haute, Ind., ALR
MILLETT, FRANK A. ('33) ..58 Federal St., Greenfield, Mass., OALR
MILLS, CHESTER R. ('34)
520 Commonwealth Ave., Boston, Mass., ALR
*MINITER, FRANCIS G. ('35) ..419 Boylston St., Boston, Mass., ALR
MINOR, H. F. ....Bank of Commerce Bldg., Memphis, Tenn., OALR
MINSKY, HENRY ('26) ....1 West 86th St., New York, N. Y., OALR
MINTON, WM. H. ('20) .....Carby Bldg., St. Joseph, Mo., OALR
MITCHELL, E. K. ('18) .710 W. Lehigh Ave., Philadelphia, Pa., ALR
MITCHELL, H. E. ('30) ....14805 Detroit Ave., Cleveland, O., ALR
*MITHOEFER, W. .....Doctors Bldg., Cincinnati, O., ALR
MITTENDORF, A. D. .....399 Park Ave., New York, N. Y., Op.
MITTENDORF, W. KEITH ('04)
115 E. 53rd St., New York, N. Y., Op.
MOEHLE, WALTER ('32) .15 Schermerhorn St., Brooklyn, N.Y., Op.
MOLL, ARTHUR M. ('32) .Powers Bldg., Grand Rapids, Mich., ALR
MONAGHAN, D. G. ('13) .....Majestic Bldg., Denver, Colo., Op.
*MONAHAN, J. A. ('13) .....181 Chestnut St., Clinton, Mass., OALR
*MONAHAN, JAMES J. ('35) ..31 S. Jordin St., Shenandoah, Pa., ALR
MONSON, LAFAYETTE ('34) .1801 Bush St., San Francisco, Calif., ALR
*MONTGOMERY, D. CAMERON ('28)
Leyser Bldg., Greenville, Miss., ALR
MOORE, G. A. ('15) .....483 Main St., Palmer, Mass., OALR
MOORE, GEORGE H. ('19)
101 Long Lane and Chestnut St., Upper Darby, Pa., OALR
*MOORE, JOHN D. ('25) ...6 Washington St., Bloomfield, N. J., OALR
*MOORE, J. HALLOCK ('35) .1050 Fifth Ave., Huntington, W.Va., ALR
MOORE, P. G. ('30) .....Republic Bldg., Cleveland, O., Op.
*MOORE, PAUL M., JR. ('30) .University Hosp., Iowa City, Ia., ALR
MOORE, R. B. ('30) .....2000 Main St., Little Rock, Ark., ALR
MOORE, ROY SEELEY ('28) ..Med. Arts Bldg., Syracuse, N. Y., ALR
*MOORE, T. K. .....Second Nat'l Bank Bldg., Akron, O., OALR
MOORE, WM. FRED'K ('22) ..255 S. 16th S., Philadelphia, Pa., ALR
MOORHEAD, ROBT. L. ('33) ..125 Remsen St., Brooklyn, N. Y., ALR
MORGAN, JAMES A. ('16) .48 Young Bldg., Honolulu, Hawaii, OALR
MORIARTY, RICHARD W. ('28)
Medical Center, White Plains, N. Y., OALR
MORLEDGE, ROY V. ('33) ...Hart Albin Bldg., Billings, Mont., ALR
MORLEY, G. A. ('30)
Crookston State Bank Bldg., Crookston, Minn., Op.
MORRIS, A. G. ('18) ....Medical Arts Bldg., Rochester, N. Y., ALR
MORRISON, WALLACE ('26) ..39 E. 50th St., New York, N. Y., ALR
*MORSE, A. W. .....507 Phoenix Bldg., Butte, Mont., OALR

*Morsman, L. Wm.('20)...Morsman Clinic, Hibbing, Minn., OALR
Morton, J. B.('24).....90 E. Warren Ave., Detroit, Mich., OALR
*Mosher, Harris P. .....828 Beacon St., Boston, Mass., ALR
Moss, Harvey E. ('20)...Professional Bldg., Phoenix, Ariz., OALR
*Moss, Olin Winn('31)....Weber Bldg., Lake Charles, La., ALR
*Motto, M. Paul('29).....Rose Bldg., Cleveland, O., Op.
Moulton, Everett C.('15)
Merchants Nat'l Bank Bldg., Ft. Smith, Ark., OALR
Moyer, John S.('34).....303 Coltart Ave., Pittsburgh, Pa., ALR
Moyle, Eugene H.(31)..1088 Park Ave., New York, N. Y., ALR
Mueller, Lillian B.('31)
Continental Bank Bldg., Indianapolis, Ind., ALR
Mullen, Clifford J.('35)
Brotherhood Bldg., Kansas City, Kon., Op.
Mulsow, J. E.('21).231 W. Wisconsin Ave., Milwaukee, Wis., OALR
Mumma, Claude S.('32)
Hotel Fort Des Moines, Des Moines, Ia., ALR
*Muncaster, S. B...Farragut Med. Bldg., Washington, D. C., Op.
Muncy, Wm. M. ('14)....162 Angell St., Providence, R. I., OALR
*Mundt, G. Henry('12)...30 N. Michigan Ave., Chicago, Ill., OALR
*Munson, F. T.('30)..2201 E. Jefferson Ave., Detroit, Mich., ALR
Murphy, A. B.('30).Med. and Dental Bldg., Everett, Wash., ALR
Murphy, Wm. E.('34).....390 Main St., Worcester, Mass., ALR
Murray, A. N.....4753 Broadway, Chicago, Ill., OALR
Murray, Harry J.('31).....53 South St., Stamford, Conn., ALR
Muskat, Irving('33)...25 E. Washington St., Chicago, Ill., OALR
Mussun, W. G.('24).....Medical Arts Bldg., Cleveland, O., ALR
Myers, Elmer A..407 Board of Trade Bldg., Superior, Wis., OALR
*Myers, Eugene Lee('26)....207 Wall Bldg., St. Louis, Mo., ALR
*Myers, John L.('20)..815 Shukert Bldg., Kansas City, Mo., OALR
Myerson, M. C.('23).....136 E. 64th St., New York, N. Y., ALR
Mysel, Philip('33).311 Commonwealth Ave., Boston, Mass., ALR

N

*Nadeau, E. G.('15).....Bellin Bldg., Green Bay, Wis., OALR
Naftzger, J. B.('17).....Davidson Bldg., Sioux City, Ia., OALR
Nash, C. Stewart('29).277 Alexander St., Rochester, N. Y., ALR
Neal, Phil H.('34).....111 E. 75th St., New York, N. Y., ALR
Nebinger, Reid('33)..Geisinger Mem. Hosp., Danville, Pa., ALR
Neeper, Edw. R.('18)
Ex. Nat'l Bank Bldg., Colorado Springs, Colo., Op.
*Neff, Eugene E.('32)
224 W. Washington Ave., Madison, Wis., Op.
Neher, Edwin M.('26).Boston Bldg., Salt Lake City, Utah, Op.
*Neller, Walter I.('33)
121 Wickham Ave., Middletown, N. Y., Op.
Nelson, Chas. F.....606 Schofield Bldg., Cleveland, O., OALR
Nelson, Leo A.('30).....4105 Live Oak St., Dallas, Texas, ALR
Nelson, Louis Allan
830 Lowry Medical Arts Bldg., St. Paul, Minn., Op.

NEMOURS, PAUL R.('29) .3142 Franklin Ave., St. Louis, Mo., ALR
NETHERCUT, G. W.('35) . .25 E. Washington St., Chicago, Ill., Op.
NEUMANN, WM. H.('28) .Sheboygan Clinic, Sheboygan, Wis., OALR
*NEW, GORDON B. . . . . .Mayo Clinic, Rochester, Minn., L
NEWCOMB, JOHN R.
410 Hume-Mansur Bldg., Indianapolis, Ind., Op.
NEWELL, WM. S.('17) .1029 Vermont Ave.,Washington, D.C., Op.
NEWLANDS, W. ALEXANDER('34) .16 Maple Pl., Ossining,N.Y., ALR
19 S. Broadway, Tarrytown,N.Y.,
NEWTON, F. H.('25) . . .907 Mercantile Bldg., Dallas, Texas, OALR
*NEWTON, MORRIS H.('34) . .Burrell Bldg., Little Falls, N. Y., Op.
NEWTON, WM. B.('31) . .201 N. Second Ave., Alpena, Mich., OALR
*NIPPE, LEONARD ('32) . . . . . .316 Michigan St., Toledo, O., OALR
NISSELSON, MAX('18) . . . . . .754 Beck St., New York, N. Y., ALR
NORCROSS, E. P.('24) . . . . . .8 S. Michigan Ave., Chicago, Ill., ALR
NORTHCUTT,THOMAS A.('26) .17 E. 38th St.,NewYork,N.Y., Op.
NORTHINGTON, PAGE('35) . .20 E. 53rd St., New York, N. Y., ALR
NORTON, ARTHUR H.('31) . .1162 Williamette, Eugene, Ore., Op.
NOVA, JULES M.('33) . . . . . .349 Adelphi St., Brooklyn, N. Y., Op.
*NOVAK, FRANK J., JR. . . . .30 N. Michigan Blvd., Chicago, Ill., ALR
*NUGENT, OSCAR B.('30) .231 W.Washington St., Chicago, Ill., Op.
NUTTER, C. F.('14) . . . . . .16 Amherst St., Nashua, N. H., Op.

O

OAST, SAMUEL P., JR.('29) .30 E. 40th St., New York, N.Y., Op.
O'BRIEN, C. S.('29) . . . . .University Hospital, Iowa City, Ia., Op.
O'BRIEN, G. R.('30) . . . .307 Sterling Place, Brooklyn, N. Y., ALR
O.BRIEN, STEVE A.('15) .1st Nat'l Bk. Bldg., Mason City, Ia., OALR
O'BRIEN, THOS. A.('33)
1727 W. Girard Ave., Philadelphia, Pa., Op.
O'CONNOR, D. F.('18) . . . . . .671 Broad St., Newark, N. J., OALR
*O'CONNOR, FRED'K J.('34) .Med. Arts Bldg., Syracuse, N. Y., ALR
*O'CONNOR, PHILIP D.('34) .30 N. Michigan Ave.,Chicago,Ill., Op.
O'CONNOR, RODERIC P.('18)
450 Sutter St., San Francisco, Calif., Op.
ODENEAL, THOMAS H.('29) . . . .21 Broadway, Beverly, Mass., OALR
also 40 Middle St., Gloucester, Mass.,
*ODOM, STANLEY G.('34)
Colon Hosp., Cristobal, Canal Zone, C. A., OALR
OHLY, JOHN H.('32) .22 Schermerhorn St., Brooklyn, N. Y., Op.
O'HORA, BERNARD A.('31) .3044 W.Grand Blvd.,Detroit,Mich., ALR
OLMSTEAD, WM. DEF. ('30)
515 Grammercy Place, Atlantic City, N. J., Op.
*OLSHO, S. L.('12) . . . . . .235 S. 15th St., Philadelphia, Pa., OALR
OPPEGAARD, C. L.('30) . .220 S. Broadway, Crookston, Minn., ALR
ORCUTT, D. C.('17) . . . . . .30 N. Michigan Ave., Chicago, Ill., Op.
O'REILLY, W. F. ('13) . . . . . .44 S. Common St., Lynn, Mass., OALR
*O'ROURKE, CARROLL('32) .347 W. Berry St., Ft. Wayne, Ind., ALR
*O'ROURKE, D. H. ('27) . . . . . .Republic Bldg., Denver,Colo., Op.
ORR, ADRIAN V. B. ('29) . . . . . .525 Welsh St., Chester, Pa., ALR

ORR, EUGENE('32).....Doctors Bldg., Nashville, Tenn., ALR
ORTON, HENRY B. ('17)...24 Commerce St., Newark, N. J., ALR
O'SULLIVAN, T. J. ('27).....10 Pine St., Portland, Me., ALR
*OTRICH, GROVER C. ('32)...Commercial Bldg., Belleville, Ill., ALR
OWSLEY, JOHN Q.('35)
Bureau of Med. & Surg., Navy Dept., Washington, D. C., ALR

P

PACE, ERRETT ('30)....City Nat'l Bank Bldg., Paducah, Ky., ALR
PACKARD, LOUIS A.('26).Haberfelde Bldg., Bakersfield Calif., OALR
PALMER, ARTHUR('26).667 Madison Ave., New York, N. Y., ALR
PALMER, BASCOM H.('28).502 Huntington Bldg., Miami, Fla., OALR
PALMER, F. E.('28).....Henderson Bldg., Sterling, Colo., OALR
*PARKER, HARRY L.('31).....88 East Ave., Lockport, N. Y., ALR
PARKER, WALTER R....David Whitney Bldg., Detroit, Mich., Op.
PARRAMORE, JAMES B.('29).St. James Bldg.,Jacksonville,Fla., ALR
PARRISH, ROBERT E...Medical Arts Bldg., San Antonio, Tex., ALR
PARRY, THAYER L.('35).....2nd Nat'l Bldg., Akron, O., ALR
PARSELL, LOUIS A.('35).Paulsen M.&D. Bldg.,Spokane,Wash., ALR
PARSONS, J. G.('14)....Crookston Clinic, Crookston, Minn., OALR
*PATTERSON, ELLEN J.('17).121 University Pl., Pittsburgh, Pa., ALR
PATTERSON, REESE W.('32).Med.Arts Bldg., Knoxville, Tenn., ALR
PATTERSON, W. E. ....Med. Arts Bldg., Minneapolis, Minn., OALR
PAUL, WM. C.('30)....Medical Arts Bldg., Tacoma, Wash., ALR
PAYNE, B. F. ('32).....35 E. 70th St., New York, N. Y., Op.
PAYNE, R. J.('26)....University Club Bldg., St. Louis, Mo., ALR
PAYNE, V. LEROY('30).Simmons Nat'l Bldg., Pine Bluff, Ark., ALR
PEARLMAN, S. J.('24)....180 N. Michigan Ave., Chicago, Ill., ALR
PEELER, C. N.('22).....106 W. 7th St., Charlotte, N. C., OALR
PEER, LYNDON A. ('31).....965 Broad St., Newark, N. J., ALR
PEERY, VANCE PRICE('29).Med. Arts Bldg., Kinston, N. C., ALR
*PEIRCE, HOWARD W.('23).David Whitney Bldg.,Detroit,Mich., ALR
*PELZ, MORT D. ('29).....Missouri Bldg., St. Louis, Mo., ALR
*PEMBER, AUBREY H. ('26)
500 W. Milwaukee St., Janesville, Wis., OALR
PENDEXTER, R. STEVENS('26)
Farragut Medical Bldg., Washington, D. C., Op.
PENTECOST, R. S.('26)....338 Bloor St., E., Toronto, Canada, OALR
*PERDUE, JAS. D.('34).Merchants Nat'l Bk. Bldg., Mobile, Ala., Op.
PERRY, C. S.('32).....40 S. Third St., Columbus, O., Op.
PERSKY, A. H.('30).....1922 Spruce St., Philadelphia, Pa., ALR
*PETER, L. C.('14).....1930 Chestnut St., Philadelphia, Pa., Op.
*PETERMAN, H. E.('12)...512 Cathedral St., Baltimore, Md., OALR
PETERSON, R. A.('20).....150 Fifth Ave., New York, N. Y., OALR
PETHERAM, CHAS. C.('31).Medical Arts Bldg.,Portland,Ore., ALR
*PFEIFFER, RAYMOND L.('35).635 W.165th St.,New York,N.Y., Op.
PHELPS,KENNETH A.('20).Med.Arts Bldg.,Minneapolis,Minn., OALR
PHILLIPS, WM. LINTON...131 Linwood Ave., Buffalo, N. Y., Op.
PINKERTON, F. J.('29).Alex. Young Bldg., Honolulu, Hawaii, OALR
PINO, RALPH H.('35)..David Whitney Bldg., Detroit, Mich., Op.

PIPER, ARTHUR SYLVESTER('28).610 S. Monroe, Enid, Okla., OALR
PITKIN, C. E.('17)....Carnegie Medical Bldg., Cleveland, O., ALR
*PLUMER, JOHN S.('28)...121 University Pl., Pittsburgh, Pa., Op.
POCHERT, ROLLAND C.('35)..Matthews Bldg., Owosso, Mich., ALR
POIRIER, GEO. H.('31)....60 Bay State Road, Boston, Mass., ALR
PONTIUS, P. J.....1831 Chestnut St., Philadelphia, Pa., Op.
POOS, E. E.('27).....Fisher Bldg., Detroit, Mich., OALR
POOS, GROVER H.('28)....607 N. Grand Ave., St. Louis, Mo., OALR
POPE, E. C.('30).....Woodward Bldg., Birmingham, Ala., ALR
PORTER, C. T.('30)..520 Commonwealth Ave., Boston, Mass., ALR
*PORTER, E. H.('12).....85 Madison St., Tiffin, O., OALR
PORTER, L. B. ('13).....199 Thayer St., Providence, R. I., OALR
PORTER, WHITNEY C.('35)....Republic Bldg., Denver, Colo., Op.
*POST, LAWRENCE T.('20).Metropolitan Bldg., St. Louis, Mo., Op.
*POST, M. HAYWARD('18).Metropolitan Bldg., St. Louis, Mo., Op.
POST, WINFRED B.('20).....304 Grant St., Carthage, Mo., OALR
*POSTLE, C. D.('30).....240 E. State St., Columbus, O., ALR
POTTER, ALLEN B.('29).....5068 Thologan, St. Louis, Mo., ALR
*POTTER, GEO. B. ('20).....Medical Arts Bldg., Omaha, Neb., OALR
POTTER, JACOB J.('35).....University Hosp., Iowa City, Ia., ALR
POTTER, L. F.....232 Walnut St., New Bedford, Mass., OALR
POTTER, PETER('18).....Motor Clinic Bldg., Butte, Mont., OALR
POTTER, W. A.('30)..7450 Grand River Ave., Detroit, Mich., ALR
*POTTER, WM. W. P.('27)...Medical Bldg., Knoxville, Tenn., OALR
POTTS, J. B. .....Medical Arts Bldg., Omaha, Neb., ALR
POWELL, LYLE S.('28)....833½ Mass. St., Lawrence, Kan., OALR
*POWERS, EVERETT('16).....305 S. Main St., Carthage, Mo., OALR
PRANGEN, AVERY DEH.('19)..Mayo Clinic, Rochester, Minn., Op.
*PRATT, FRED J.('12).Phys. & Surg. Bldg., Minneapolis, Minn., OALR
PRENDENGAST, D. A. .....Medical Arts Bldg., Cleveland, O., OALR
PRESSMAN, JOEL J.('35).1917 Wilshire Blvd., Los Angeles, Calif., ALR
*PRESTON, H. GRANT('30).Prof'snal Bldg., Harrisonburg, Va., ALR
PRICE, JOHN B.('29).....824 DeKalb St., Norristown, Pa., ALR
PRINCE, ALEXANDER L.('28)...36 Pearl St., Hartford, Conn., Op.
*PROETZ, A. W.('23).....Beaumont Bldg., St. Louis, Mo., ALR
PROUD, WILLARD C.('20).Phys.&Surg. Bldg., St. Joseph, Mo., OALR
PROUT, A. W.('20).....380 E. Town St., Columbus, O., OALR
PUTNAM, F. I.....1401 Prospect Ave., Cleveland, O., OALR
PYLE, WALLACE('15)....15 Exchange Pl., Jersey City, N. J., OALR

Q

QUALLS, HARLEY W.('28).Exchange Bldg., Memphis, Tenn., OALR
QUINBY, WM. O'G.('19).....14 James St., Newark, N. J., OALR
QUINCY, J. E.('27).270 Commonwealth Ave., Boston, Mass., ALR
QUIRING, WALTER O.('35).100 First West, Hutchinson, Kan., ALR
*QUITTNER, S. S.('15).....10515 Carnegie Ave., Cleveland, O., OALR

R

*RALLS, C. T.('16).....1st Nat'l Bank Bldg., Winfield, Kan., OALR
RALSTON, FRANK L.('33).Sacajawea Annex, La Grand, Ore., ALR

RALSTON, WM. W.(.18)..1300 Walker Ave., Houston, Tex., Op.
RAMSEY, G. STUART('27).1496 Mountain St., Montreal, Can., Op.
*RANDALL, HIRAM('28).....81 Main St., Binghamton, N. Y., ALR
*RAVDIN, B. D. ....Central Union Bk. Bldg., Evansville, Ind., OALR
*RAY, VICTOR, JR. ('32).....Carew Tower, Cincinnati, O., Op.
*REAVES, ROBERT GRISHAM('28)
422 W. Cumberland Ave., Knoxville, Tenn., OALR
REAVES, W. P.('13)
Reaves Infirm., 117 W. Sycamore St., Greensboro, N. C., OALR
RECTOR, A. E.('15)..103 W. Sycamore Ave., Appleton, Wis., OALR
*REED, CHAS. L.('33).....Jenkins Bldg., Pittsburgh, Pa., Op.
*REED, WM. W.('30).....Mills Bldg., Topeka, Kan., ALR
*REEDER, J. E. ('16).....Davidson Bldg., Sioux City, Ia., OALR
*REESE, ALGERNON B.('28)..73 E. 71st St., New York, N. Y., Op.
REESE, WARREN S.('21)....230 S. 21st St., Philadelphia, Pa., Op.
*REGAN, JAS. J.('32).520 Commonwealth Ave., Boston, Mass., Op.
REICHE, O. C.('18).....Markle Bank Bldg., Hazleton, Pa., Op.
*REID, H. W.('30).....Doctors Bldg., Cincinnati, O., Op.
REIM, HUGO.....417 Metropolitan Bldg., St. Louis, Mo., Op.
REIMERS, R. S.('35).....613 Eighth St., Fort Madison, Ia., ALR
REINEKE, GEO. F.('26).....Box 137, New Ulm, Minn., OALR
RENAUD, G. L.....510 Joseph Campan Ave., Detroit, Mich., OALR
*REPASS, R. E.('20).....835 Lincoln Rd., Miami Beach, Fla., ALR
REULING, F. H.('24).....Black Bldg., Waterloo, Ia., OALR
REYNOLDS, J. S.('18).Medical Arts Bldg., Minneapolis, Minn., OALR
*RHEIN, ALFRED ED.('31)..Tribune Bldg., Terre Haute, Ind., ALR
RHODE, SOLON LAFAYETTE('28).238 N. 6th St., Reading, Pa., OALR
RICE, CLARENCE PRENTICE('22).....Breckenridge, Minn., OALR
RICE, G. B. ('18).....99 Bay State Road, Boston, Mass., ALR
RICE, JOHN E. ('24).....390 Main St., Worcester, Mass., OALR
RICHARDS, L. G.('24)....319 Longwood Ave., Boston, Mass., ALR
*RICHARDSON, J. J.('29)
Cook Hosp. and Beall Clinic, Fort Worth, Texas, ALR
RICHARDSON, SHALER('25).111 W. Adams, Jacksonville, Fla., Op.
RICHIE, L. T.('18).....McCormick Bldg., Trinidad, Colo., OALR
RICHMAN, FRANCES('35)...522 Ocean Ave., Brooklyn, N. Y., Op.
RICKER, WM. G. ('33).....33 Main St., St. Johnsbury, Vt., Op.
RIDDELL, MALCOLM M.('35)...112½ Front St., Fremont, O., ALR
*RIDPATH, ROBERT F.('14)
N.E. cor. 18th and Chestnut Sts., Philadelphia, Pa., LR
RIEGER, W. H.('24).....Hanna Bldg., Cleveland, O., ALR
RIGG, J. P.('30).....217½ N. Pine St., Grand Island, Nebr., ALR
RIGLEY, ED. L.('34).....Sherland Bldg., South Bend, Ind., ALR
*RIKER, AARON D.('34)....1012 Riker Bldg., Pontiac, Mich., Op.
*RIMER, FRANK H.('35).7026 Jenkins Arcade, Pittsburgh, Pa., ALR
RINGUEBERG, EUGENE N. S.('25).13 Main St.,Lockport,N.Y., Op.
RISCH, OTTO CARL('35)....104 E. 40th St., New York, N. Y., ALR
RISDON, E. F.('24)....Medical Arts Bldg., Toronto, Canada, ALR
*RISSER, EDWARD D.('28)..310 Choate Bldg., Winona, Minn., OALR
RIWCHUN, MEYER H.('34).468 Delaware Ave., Buffalo,N.Y., Op.

ROBB, MAYO ('29) . . . . . Professional Bldg., Phoenix, Ariz., ALR
*ROBERTS, S. E. ('16) . . . Professional Bldg., Kansas City, Mo., ALR
*ROBERTSON, EDW. N. ('18) . New Caldwell Bldg., Concordia, Kan., OALR
ROBERTSON, F. D. . . . . Ashton Bldg., Grand Rapids, Mich., OALR
ROBINSON, H. T. ('16) . . . . . Medical Bldg., Cumberland, Md., OALR
ROBINSON, R. E. ('20) . . 205½ E. Bremer Ave., Waverly, Ia., OALR
ROBISON, J. T. ('30) . . . . . Norwood Bldg., Austin, Texas, ALR
ROGERS, J. T. . . . . Medical Arts Bldg., Montreal, Can., ALR
ROGERS, ROBERT M. ('28) . 680 St. John's Pl., Brooklyn, N. Y., Op.
ROMMEL, JOHN C. ('20) . 3701 N. Broad St., Philadelphia, Pa., Op.
ROMONEK, PHILIP L. ('29) . . . Med. Arts Bldg., Omaha, Nebr., OALR
RONES, BENJAMIN ('35) . 1610 Eye St. N.W., Washington, D.C., Op.
*ROOKER, A. M. ('20) . . . . . Gluck Bldg., Niagara Falls, N. Y., ALR
ROSEBROUGH, F. H. ('29) . . . . Moore Bldg., San Antonio, Tex., Op.
*ROSENBAUM, J. ('15)
1396 St. Catherine's St. W., Montreal, Can., Op.
ROSENBAUM, MORRIS ('29) . 61 W. 88th St., New York, N. Y., Op.
ROSENBERGER, H. C. ('29) . 10515 Carnegie Ave., Cleveland, O., ALR
ROSENBLUTH, MICHAEL ('31)
1018 E. 163rd St., New York, N. Y., ALR
ROTH, ALBERT W. ('20) . . . . Medical Arts Bldg., Tulsa, Okla., OALR
ROTHSCHILD, HAROLD J. . . . . Lowry Bldg., St. Paul, Minn., OALR
*ROTT, O. M. ('15) . . Paulsen M. and D. Bldg., Spokane, Wash., OALR
ROW, D. HAMILTON ('35) . . 23 E. Ohio St., Indianapolis, Ind., Op.
ROWLAND, W. D. ('23) . 84 Commonwealth Ave., Boston, Mass., Op.
RUBENDALL, C. ('15) . . . . . Medical Arts Bldg., Omaha, Nebr., OALR
*RUBENSTEIN, A. HARRY ('30) . Med. Arts Bldg., Syracuse, N.Y., ALR
RUCKER, C. W. ('29) . . . Med. Arts Bldg., Minneapolis, Minn., Op.
*RUCKER, E. W., JR. ('16)
926 Woodward Bldg., Birmingham, Ala., ALR
RUDOLPHY, J. B. ('29) . . . . . 1924 Pine St., Philadelphia, Pa., Op.
*RUEDEMANN, A. D. ('29)
Cleveland Clinic, Euclid Ave. and 93rd St., Cleveland, O., Op.
*RUNK, R. L. ('29)
Wash. Sanit. and Hosp., Takoma Pk., Washington, D. C., ALR
RUSSELL, J. A. MERLE ('22) . . . . . 813 Sassafras St., Erie, Pa., OALR
RUSSELL, ROLAND D. ('31) . 55 E. Washington St., Chicago, Ill., ALR
*RUSSELL, R. E. ('24) . . . . . 606 Commercial St., Waterloo, Ia., OALR
*RUTHERFORD, C. W. ('25)
Hume-Mansur Bldg., Indianapolis, Ind., Op.
RYAN, ED. R. ('30) . . . . . Wells Bldg., Milwaukee, Wis., Op.
RYAN, L. M. ('27) . . . . . Wall Bldg., St. Louis, Mo., ALR
*RYCHENER, RALPH O. ('28) . . . 130 Madison, Memphis, Tenn., Op.
RYDER, D. R. . . . . 151 Rock St., Fall River, Mass., ALR
*RYERSON, F. L. ('25) . . . . . Fine Arts Bldg., Detroit, Mich., OALR

S

SACCO, ANTHONY G. ('20)
440 New York Ave., Union City, N. J., OALR
SACHS, BENJAMIN ('35) . . . . . 483 Beacon St., Boston, Mass., Op.

SACKIN, DAVID ('33)
"St. Moritz," 50 Central Park South, New York, N. Y., ALR
*SALINGER, SAMUEL ('20) . 25 E. Washington St., Chicago, Ill., ALR
SALLANDER, F. W. ('19) . New Orpheum Bldg., Sioux City, Ia., OALR
*SAMUELS, BERNARD ('29) . . . 57 W. 57th St., New York, N. Y., Op.
*SANDERS, JOHN GILLIS ('29)
Merchants Nat'l Bank Bldg., Mobile, Ala., ALR
SANDERS, SAM H. ('32) . . 248 Madison Ave., Memphis, Tenn., ALR
SANDERSON, DAVID D. ('17) . . 914 Stuart Bldg., Lincoln, Neb., OALR
SANOR, DANIEL G., JR. ('28) . . 206 E. State St., Columbus, O., ALR
SARTAIN, PAUL J. ('21) . . . 2006 Walnut St., Philadelphia, Pa., Op.
*SAVERY, C. E. ('22) . . . . 206 Sherland Bldg., South Bend, Ind., OALR
*SAWYER, LEROY L., JR. ('34)
Farragut Med. Bldg., Washington, D. C., ALR
SAYAD, WM. Y. ('32) . . Harvey Bldg., West Palm Beach, Fla., ALR
SCAL, J. COLEMAN ('26) . . . 27 W. 96th St., New York, N. Y., ALR
*SCALES, H. L. ('20) . . . . Rorabaugh Bldg., Hutchinson, Kan., OALR
SCALES, J. L. ('15) . . . . . Medical Arts Bldg., Shreveport, La., OALR
SCARLETT, HUNTER W. ('18) . 230 S. 21st St., Philadelphia, Pa., Op.
SCARNEY, HERMAN DAVID ('33)
2501 W. Grand Blvd., Detroit, Mich., Op.
*SCHALL, LEROY A. ('27)
270 Commonwealth Ave., Boston, Mass., ALR
SCHAPPERT, N. LOUIS ('16)
59 S. Washington St., Wilkes-Barre, Pa., OALR
SCHECHTER, SOLOMON ('35)
1749 Grand Concourse, New York, N. Y., ALR
SCHEER, HENRY M. ('32) . 522 West End Ave., New York, N. Y., ALR
SCHENCK, C. P. ('20) . . . . Med. Arts Bldg., Fort Worth, Tex., OALR
SCHIEFELBEIN, HARRY T. ('31)
Stevens Clinic Hospital, Welch, W. Va., Op.
SCHILLER, A. NOAH ('18) . 225 Central Pk. W., New York, N. Y., ALR
SCHILLINGER, RAPHAEL ('34) . . 50 Plaza St., Brooklyn, N. Y., ALR
SCHLANSER, MAJOR A. E. ('21)
Letterman General Hosp., San Francisco, Calif., OALR
SCHLEGEL, HERMAN T. ('16) . . 501 Third St., Wausau, Wis., OALR
SCHLINDWEIN, G. WM. . . . . 138 W. 9th St., Erie, Pa., OALR
SCHLIVEK, KAUFMAN ('25) . 1016 Fifth Ave., New York, N. Y., Op.
SCHLUER, E. P. ('30) . . . . . 508 N. Grand Ave., St. Louis, Mo., ALR
SCHMIDT, HERBERT G. ('34)
238 W. Wisconsin Ave., Milwaukee, Wis., ALR
SCHMIDT, OTTO V. M. ('32) . 111 E. 61st St., New York, N. Y., ALR
SCHNECK, NATHAN ('31) . . Commerce Blk., Manitowoc, Wis., ALR
SCHNEIDER, ANTON S. ('28) . . . 56 Court St., Plattsburg, N. Y., Op. A
*SCHOENBERG, MARK J. ('32) . 667 Madison Ave., New York, N. Y., Op.
*SCHOLZ, R. P. ('16) . . . . . Metropolitan Bldg., St. Louis, Mo., ALR
SCHRANT, J. H. ('16) . . . . . 14 W. 1st St., Hutchinson, Kan., OALR
*SCHUBERT, CLARENCE K. ('33) . 1 S. Pinckney, Madison, Wis., Op.
SCHUGT, HENRY PAUL ('33) . 30 E. 40th St., New York, N. Y., ALR
SCHULZE, E. C. ('26) . . . 504 First Nat'l Bldg., Del Rio, Tex., OALR

SCHUSTER, FRANK P.('27) .1st Nat'l Bk. Bldg., El Paso, Tex., OALR
SCHUSTER, S. A.('24) . . . . .1st Nat'l Bk. Bldg., El Paso, Tex., ALR
SCHWARTZ, A. A.('27) . . . . . .7 W. 71st St., New York, N. Y., ALR
SCHWARTZ, FRED'K O.('19)
508 N. Grand Blvd., St. Louis, Mo., Op.
*SCHWARTZ, LEO, JR.('35) . . .1 W. 73rd St., New York, N. Y., ALR
SCHWARTZ, V. J.('29) . .Med. Arts Bldg., Minneapolis, Minn., ALR
SCHWARTZ, WM. A.('28) .Professional Bldg., Phoenix, Ariz., OALR
*SCIARRETTA, SYLVIO A.('31) .55 E. Washington St.,Chicago,Ill., ALR
*SCOTT, MERLE E. ('26) . .1st Nat'l Bank Bldg., Massillon, O., OALR
*SCROGGS, J. J.('21) . . . . . .218 E. Third St., Beaver, Pa., Op.A
*SEARCY, H. B. ('12) . . . . . .2501 Sixth St., Tuscaloosa, Ala., OALR
*SEBERT, L. J.('30) . . . . .Medical Arts Bldg., Toronto, Canada, Op.
*SECOY, F. L. ('22) . . . . . .301 Davidson Bldg., Sioux City, Ia., OALR
SECOY, HARRY R.('35) . . . . . .Everett Clinic, Everett, Wash., ALR
SEDGWICK, O. W.('17) . . . . . .Ionia, Mich., OALR
SEDWICK, W. A. ('16) . . . . . .Republic Bldg., Denver, Colo., Op.
SEEGMAN, SIMON('17) . . . . . .Medical Bldg., Pittsburgh, Pa., ALR
SEELYE, W. K.('20) . . . . . .1215 Fourth Ave., Seattle, Wash., OALR
SEGAL, SAMUEL, JR.('33) . .1562 Main St., Springfield, Mass., ALR
*SEIDEL, U. D.('34) . . . . . .2nd Nat'l Bank Bldg., Akron, O., ALR
SELIGSTEIN, MILTON B.('26)
801 Union and Planters Bk. Bldg., Memphis, Tenn., OALR
SELINGER, ELIAS('35) . . . .55 E. Washington St., Chicago, Ill., Op.
SELINGER, SAMUEL('33) .413 16th St., West New York, N. J., OALR
SELLERS, L. M. . . . . .Medical Arts Bldg., Dallas, Texas, ALR
*SELTZ, SAMUEL('31) . . .Provident Bank Bldg., Cincinnati, O., ALR
*SEMENOV, HERMAN('35) .500 S. Lucas Ave.,LosAngeles,Calif., ALR
SENER, WALTER J.('20) . .Physicians Bldg., Philadelphia, Pa., Op.
SENSENEY, EUGENE T.('28) .316 Lister Bldg., St., Louis, Mo., ALR
SETTEL, NATHAN('31) . . . .10 W. 82nd St., New York, N. Y., ALR
SEWARD, JACKSON A.('31) . .115 E. 61st St., New York, N.Y., ALR
SEYDELL, E. M.('16) .1030 1st Nat'l Bk. Bldg., Wichita, Kan., ALR
SHAFER, J. J.('13) . . . . . .Starks Bldg., Louisville, Ky., OALR
SHAHAN, W. E.('17) . . . .Metropolitan Bldg., St. Louis, Mo., Op.
SHANKLIN, E. M. . . . . .575 Hohman St., Hammond, Ind., OALR
SHANNON, C. E. G. . . . .1930 Chestnut St., Philadelphia, Pa., Op.
SHANNON, CLARENCE WM.('31)
New 4th and Pike Bldg., Seattle, Wash., Op.
SHAPERO, I. M.('19) . . . .289 Alexander St., Rochester, N. Y., OALR
SHAPIRA, THEO. M.('34) .58 E. Washington St., Chicago, Ill., Op.
SHAPIRO, S. L.('30) . . . . . .185 N. Wabash Ave., Chicago, Ill., ALR
SHARP, BENJ. S. ('26) . . . .339 Thayer St., Providence, R. I., OALR
SHARRETT, GEO. O.('21) . .119 Bedford St., Cumberland, Md., OALR
SHAWHAN, GLENN E.('13) .Dewey Scales Bldg.,Nampa,Ida., OALR
*SHEA, J. J. ('16) . . . . . .1018 Madison Ave., Memphis, Tenn., ALR
SHEAHAN, WM. L.('26) . .59 College St., New Haven, Conn., OALR
SHELLMAN, J. L.('17) .Lowry Med. Arts Bldg.,St.Paul,Minn., OALR
SHEMELEY, WM. G.('19) . . . . .7 Haddon Ave., Camden, N. J., OALR

SHERMAN, MAJ. A. ED.('13), M.C., U.S.A., N.Y. Port of Embarkation, Off. Port Surg., 58th St. & 1st Ave., Brooklyn, N.Y., OALR
*SHERMAN, A. RUSSELL('33)...671 Broad St., Newark, N. J., Op.
SHERMAN, ELBERT S.('12)....671 Broad St., Newark, N. J., Op.
*SHERMAN, R. NEWTON('34).Phoenix Bldg., Bay City, Mich., OALR
SHIELDS, J. M.('15)...264 Metropolitan Bldg., Denver, Colo., Op.
SHIELDS, LAWRENCE.....Allen Bldg., Xenia, O., ALR
SHIPLEY, JOHN LEROY('33), care Bureau of Medicine and Surgery, Navy Dept., Washington, D. C., OALR
SHIPMAN, JAMES S.('35).....542 Cooper St., Camden, N. J., Op.
SHIRAS, HOWARD H.('20).624 Guardian Bldg., Cleveland, O., Op.
*SHIREY, O. M.('25).....Osborn Bldg., Cleveland, O., ALR
SHOEMAKER, FERDINAND('20) U. S. Veterans' Hospital, Excelsior Springs, Mo., OALR
SHOEMAKER, S. A.('17).....30 E. Church St., Orlando, Fla., OALR
SHOPE, PIERCE('32).....511 Cooper St., Camden, N. J., Op.
SHULTS, E. J. G.('20).....Haines Bldg., Eldorado, Kan., OALR
SHUMAN, G. H.('17).....Park Bldg., Pittsburgh, Pa., Op.
SHUMATE, D. L.....500 Argyle Bldg., Kansas City, Mo., OALR
*SHUSTER, B. H.('29)...1930 Chestnut St., Philadelphia, Pa., ALR
SHUTE, D. K. ....1727 DeSales St., N.W., Washington, D. C., Op.
SIDWELL, C. E.('29).....608 Fourth Ave., Longmont, Colo., ALR
*SIEGEL, FRANCIS X.('22) 610 Union Central Life Bldg., Cincinnati, O., OALR
*SILBERNAGEL, C. E.('18).....247 E. State St., Columbus, O., OALR
SILVA, RAFAEL('32).....Av. Insurgentes 195, Mexico, D. F., Op.
SIMMONS, RALPH H. (Major M. C.) ('34) Army Medical Center, Washington, D. C., ALR
SIMON, EMANUEL('32).....238 Lark St., Albany, N. Y., ALR
SIMON, F. C.('29).....914 Arcade Bldg., St. Louis, Mo., ALR
SIMONDS, O. F. .....Hanna Bldg., Cleveland, O., Op.
SIMONS, N. S.('30).....MacCornack Clinic, Whitehall, Wis., ALR
*SIMPSON, GEO. V.('31) 1710 R. I. Ave., N.W., Washington, D. C., Op.
*SIMPSON, H. LEE('21)..David Whitney Bldg., Detroit, Mich., ALR
*SIMPSON, JOHN REID('27)..Med. Arts Bldg., Pittsburgh, Pa., ALR
*SIMPSON, M. B.('30)...Professional Bldg., Kansas City, Mo., ALR
SINAMARK, ANDREW('28)..205 Power Bldg., Hibbing, Minn., OALR
SITCHEVSKA, OLGA L.('32).160 W. 87th St., NewYork, N.Y., Op.
SKILLERN, S. R., JR.('25)....1734 Pine St., Philadelphia, Pa., ALR
SKIRBALL, JOSEPH J.('27) 353 Commonwealth Ave., Boston, Mass., Op.
*SLACK, WALTER K.('34).....Eddy Bldg., Saginaw, Mich., ALR
SLOAN, HENRY L.('19).....106 W. 7th St., Charlotte, N. C., Op.
SLOANE, H. O.('30).....1717 Pine St., Philadelphia, Pa., Op.
*SLOTKIN, H. B.('32).....311 S. 20th St., Philadelphia, Pa., ALR
SLY, JOHN L.('33).....382 Springfield Ave., Summit, N. J., ALR
SMIT, WM. M.('28)..5285 Washington Blvd., St. Louis, Mo., ALR
SMITH, ARTHUR E. ..Medical Arts Bldg., Minneapolis, Minn., OALR

SMITH, CARLIE SOUTER('27)
214 North Jefferson Ave., Springfield, Mo., OALR
SMITH, CARROLL L.('29)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
SMITH, D. S.('18).....State Bank Bldg., La Crosse, Wis., OALR
SMITH, E. TERRY.....36 Pearl St., Hartford, Conn., Op.
*SMITH, FERRIS('33).Brodgett Med. Bldg.,Grand Rapids,Mich., ALR
SMITH, GEORGE B. ('14).....Harbin Hospital, Rome, Ga., OALR
SMITH, H. A.('25).....226 N. Greenleaf St., Whittier, Calif., OALR
SMITH, JAS. M.('31).....123 E. 53rd St., New York, N. Y., ALR
SMITH, J. M.....134½ N. Patterson St., Valdosta, Ga., OALR
SMITH, R. C.('18)...Board of Trade Bldg., Superior, Wis., OALR
SMITH, R. N.('29).....Medical Arts Bldg., Tulsa, Okla., ALR
SMITH, VICTOR C.('12).American Bk. Bldg.,New Orleans,La., Op.
*SMITH, WM. L.('30).Med. Arts Bldg., Salt Lake City, Utah, Op.
*SMITH, WINDSOR R.('33)..67 Front St., Binghamton, N. Y., ALR
*SNAPP, CARL F.('28).604 Med. Arts Bldg.,Grand Rapids,Mich., ALR
SNEED, CARL M. .....Guitar Bldg., Columbia, Mo., OALR
SNELL, ALBERT C.('25).53 S. Fitzhugh St., Rochester, N. Y., Op.
SNELL, GEO. C.('33).....Medical Arts Bldg., Toronto, Can., ALR
SNELLING, P. W. ('31).....179 Allyn St., Hartford, Conn., ALR
SOMBERG, J. S.('23).....470 Park Ave., New York, N. Y., Op.
also 127 Overdale Road, Greenhaven, Rye, N. Y.
*SONNENSCHEIN, ROBERT('15)
180 N. Michigan Ave., Chicago, Ill., ALR
*SOPER, GAIL R. ('33).....636 Church St., Evanston, Ill., Op.
SPAETH, EDMUND B.('28).1930 Chestnut St.,Philadelphia,Pa., Op.
SPAKE, LAVERNE B.('20).Brotherhood Bldg.,Kansas City,Kan., ALR
SPARBER, HERMAN('35)...4701 15th Ave., Brooklyn, N. Y., Op.
SPENCER, GEO. A.('28)...1025 Ninth St., Sacramento, Calif., OALR
SPENCER, S. C.('29).....Professional Bldg., Waco, Texas, Op.
SPENCER, W. H.('18)
1830 S. Rittenhouse Sq., Philadelphia, Pa., ALR
SPENGLER, JOHN ARTHUR('16).423 S. Main St., Geneva, N.Y., Op.
SPIESMAN, I. G.('29).....715 Lake St., Oak Park, Ill., ALR
SPITZE, E. C.('16).....Murphy Bldg., East St. Louis, Ill., Op.
SPROULE, RALPH P.('28)
208 E. Wisconsin Ave., Milwaukee, Wis., OALR
*ST. CLAIR, C. T.('30)
Corner Bland and Ramsey Sts., Bluefield, W. Va., ALR
STAHLMAN, FRED C.('22)Commercial Bldg., Waynesburg, Pa., OALR
*STANFORD, J. B. ('16)...899 Madison Ave., Memphis, Tenn., OALR
*STANLEY, C. R.('30)....508 Tenth St., Worthington, Minn., ALR
*STANTON, HARMON LAMONTTE ('28)
Central Union Bank Bldg., Evansville, Ind., OALR
STARK, JESSE B.('26).....45 Park Ave., New York, N. Y., Op.
STARK, WM. B.('27).....Med. Arts Bldg., Toronto, Canada, ALR

STAUFFER, F. LEAVER('34)
707 Med. Arts Bldg., Salt Lake City, Utah, ALR
STAUFFER, FRED ('12). Med. Arts Bldg., Salt Lake City, Utah, OALR
*STEELE, WILLARD('28)...721 Broad St., Chattanooga, Tenn., OALR
STEIM, J. M.('14). 1st Nat'l Bk. Bldg., New Kensington, Pa., Op.
STEINBUGLER, WM. F. C.('35). 515 Ocean Ave., Brooklyn, N.Y., Op.
STEINFELD, A. L.('22).....125 Fifteenth St., Toledo, O., OALR
*STERN, MILTON J.('35). 1st Nat'l Bank Bldg., Lexington, Ky., Op.
STEVENSON, WALTER('15).....W. C. W. Bldg., Quincy, Ill., OALR
*STEVENSON, WM. W.('31).....Kresge Bldg., Flint, Mich., ALR
*STEWART, WALTER E.('20). Tribune Bldg., Terre Haute, Ind., OALR
STILES, PORTER('32).....Med. Arts Bldg., Birmingham, Ala., ALR
STIMSON, GEORGE W.('19).....Mahoning Bldg., Warren, O., OALR
STINE, GEO. H.('32)...Burns Bldg., Colorado Springs, Colo., Op.
STINSON, WM. D.('28). 805 Med. Arts Bldg., Memphis, Tenn., ALR
*STITT, H. L.('27).....656 Doctors Bldg., Cincinnati, O., ALR
STOKES, HARRY B.('34).....130 S. Scott St., Tuscon, Ariz., ALR
STOKES, WM. H. ('26)....Medical Arts Bldg., Omaha, Neb., Op.
*STOLL, K. LUDWIG.....19 W. 7th St., Cincinnati, O., OALR
STONE, H. B.('30).....Medical Arts Bldg., Roanoke, Va., ALR
STONE, LESLIE O.('35), Lieut-Comdr. (M.C.), U. S. Navy,
U. S. Naval Hospital, Great Lakes, Ill., ALR
STONECYPHER, D. D.('30). 107 S. 9th St., Nebraska City, Neb., ALR
STOTTER, ARTHUR L.('16)..1148 Euclid Ave., Cleveland, O., OALR
STOUT, PHILIP S. .....269 S. 19th St., Philadelphia, Pa., ALR
*STOUTENBOROUGH, WM. A.('27). 21 E. State St., Columbus, O., OALR
*STRAATSMA, CLARENCE R.('32)
899 Park Ave., New York, N. Y., ALR
STRAUSS, J. F.('25).....104 S. Michigan Blvd., Chicago, Ill., ALR
STREIT, AUGUST J.('31).....Fisk Bldg., Amarillo, Texas, ALR
STRICKLER, D. A.('19).....1520 York St., Denver, Colo., Op.
STRONG, J. C.('15).....810 Harrison Ave., Leadville, Colo., OALR
STROUD, EDWARD FRANK('26)
Medical Professional Bldg., Corpus Christi, Texas, ALR
STROUT, G. ELMER('21). 1009 Nicollet Ave., Minneapolis, Minn., OALR
*STUEBER, PAUL J. ('17).....209 W. North St., Lima, O., OALR
STURGES, LEIGH F.('18)...121 E. 60th St., New York, N. Y., ALR
SULLIVAN, J. J., JR. ('23)
228 Scranton Life Bldg., Scranton, Pa., ALR
*SULLIVAN, JOSEPH A.('33). 170 St. George St., Toronto, Can., ALR
*SULLIVAN, ROBT. E.('32)....Doctors Bldg., Nashville, Tenn., Op.
SULZER, C. A.('07).....327 E. State St., Columbus, O., OALR
*SULZMAN, FRANK MALCOLM('22). 1831 5th Ave., Troy, N.Y., OALR
*SUMMERS, WM. S. ('27)
1613 David Whitney Bldg., Detroit, Mich., OALR
SUTHERLAND, J. M.('29)
8-257 General Motors Bldg., Detroit, Mich., ALR
SWAB, CHAS. MARION('28)..Med. Arts Bldg., Omaha, Nebr., Op.

SWAN, C. J.('11) . . . . . .636 Church St., Evanston, Ill., OALR
*SWANN, CECIL C.('32) . .706 Flatiron Bldg., Asheville, N. C., ALR
SWERDFEGER, E. B.('27) . . . . . 1763 Gilpin St., Denver, Colo., ALR
SWETMAN, C. R. K.('15) . . . Masonic Temple, Prescott, Ariz., ALR
*SWING, FRANK ULMER('35) . . . Doctors Bldg., Cincinnati, O., ALR

T

*TANGEMAN, H. F.('19)
606 Union Central Bldg., Cincinnati, O., ALR
*TANNER, JAS. W.('32) . .314 E. Grand Ave., Eau Claire, Wis., ALR
TAQUINO, GEO. J.('20) . 208 Audubon Bldg., New Orleans, La., ALR
TARUN, WILLIAM . . . . .104 W. Madison St., Baltimore, Md., Op.
TASSMAN, ISAAC C.('29) . Med. Arts Bldg., Philadelphia, Pa., Op.
TAYLOR, CHARLES B.('21) . . . . .118 E. 2nd St., Ottumwa, Ia., ALR
TAYLOR, EDGAR M.('31) . Medical Arts Bldg., Portland, Ore., Op.
TAYLOR, H. MARSHALL('35) . 111 W. Adams, Jacksonville, Fla., ALR
TAYLOR, JOSEPH W.('34) . . Citizens Bank Bldg., Tampa, Fla., ALR
TAYLOR, M. C. ('17) . . . . . 1204 May Bldg., Pittsburgh, Pa., Op.
TEALL, RAYMOND E.('28) . Professional Bldg.,Kansas City,Mo., ALR
TEBBUTT, H. K. ('30) . . . . . 240 State St., Albany, N. Y., ALR
TERRY, THEODORE L.('32) . . . 128 Newbury St., Boston, Mass., Op.
*DVORAK-THEOBALD, GEORGIANA('29)
715 Lake St., Oak Park, Ill., Op.
THEOBALD, JOHN J.'29) . . 58 E. Washington St., Chicago, Ill., ALR
*THEOBALD, W. H.('30) . . 307 N. Michigan Ave., Chicago, Ill., ALR
THIGPEN, C. A. . . . . 401 S. Court St., Montgomery, Ala., OALR
THOMAS, C. D.('14) . . . Central Nat'l Bank Bldg., Peoria, Ill., OALR
*THOMAS, FRANCIS W.('16) . . 327 E. State St., Columbus, O., OALR
THOMAS, H. G.(26) . . . . . 411 Thirtieth St., Oakland, Calif., OALR
THOMAS, JAMES W.('28) . . . . . Sharp Bldg., Lincoln, Nebr., OALR
THOMAS, W. MAXWELL('35) . Medical Arts Bldg., Dallas, Tex., Op.
THOMASHEFSKY, MILTON('30)
135 Eastern Parkway, Brooklyn, N. Y., ALR
THOMASON, H. E. . . . . Professional Bldg., Kansas City, Mo., ALR
THOMPSON, HOWARD E.('28) . . . 1100 Main St., Dubuque, Ia., OALR
THOMPSON, PETER H.('14)
308 Commonwealth Ave., Boston, Mass., Op.
*THORPE, HARVEY ED.('31)
8075 Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*THYGESON, PHILLIPS('34)
Dept. Ophth., University Hosp., Iowa City, Ia., Op.
*TILDERQUIST, D. L.('25) . Medical Arts Bldg., Duluth, Minn., OALR
TILLOTSON, R. S.('30)
Medico-Dental Bldg., Sacramento, Calif., ALR
*TINDAL, JOHN M. ('33)
4738 W. Washington Blvd., Chicago, Ill., OALR
TINGLEY, LOUISA PAINE('13) . .9 Mass. Ave., Boston, Mass., Op.
Also 171 Westminster St., Providence, R. I.
TIPPIN, ERNEST E.('34) . 1st Nat'l Bank Bldg., Wichita, Kan., ALR

TITSWORTH, GUY .....111 W. 4th St., Sedalia, Mo., OALR
TOBEY, GEO. L., JR. . .270 Commonwealth Ave., Boston, Mass., ALR
TODD, GROVER C.('22) . .6133 Jenkins Arcade, Pittsburgh, Pa., ALR
*TOLAN, THOS. L.('33) . . .1238 Wells Bldg., Milwaukee, Wis., ALR
*TOMASSENE, R. A.('23) .1144 Market St., Wheeling, W. Va., OALR
TOMB, E. H.('30) . . . . .46 Lexington St., Framingham, Mass., ALR
*TOMLIN, W. S.('12) . Hume-Mansur Bldg., Indianapolis, Ind., ALR
TOOKER, C. W. ('17) . . . . . Carleton Bldg., St. Louis, Mo., Op.
TOOT, J. F.('30) . . . . . First Nat'l Bank Bldg., Canton, O., Op.
*TOWNES, C. DWIGHT('32) . . . . Heyburn Bldg., Louisville, Ky., Op.
TOWNSEND, JOHN F.('23) .85 Wentworth St.,Charleston,S.C., OALR
TOWSON, CHAS. E.('32)
Germantown Prof. Bldg., Philadelphia, Pa., ALR
*TRAUGH, GEO. H.('33) .309 Cleveland Ave., Fairmont, W.Va., ALR
TREMBLE, GEORGE ED. ('32)
1390 Sherbrooke St., W., Montreal, Can., ALR
TRIBLE, G. B. ('20) . .1801 Eye St., N.W., Washington, D. C., OALR
TRIMBLE, CLARENCE S.('20) . . .Gazette Bldg., Emporia, Kan., OALR
*TRIPP, LEROY R. . . . . Security Bldg., Sioux City, Ia., OALR
TROTTER, HOMER A.('32) . . . . . 40 North St., Buffalo, N. Y., ALR
*TUCKER, GABRIEL('23) . . .326 S. 19th St., Philadelphia, Pa., B
TUCKERMAN, W. C.('11) . . .733 Osborn Bldg., Cleveland, O., Op.
TURNBULL, F. M.('16) . Wilshire Med. Bldg.,Los Angeles,Calif., ALR
TURNER, H. H.('14) . . . . . Clark Bldg., Pittsburgh, Pa., Op.
TURNLEY, WM. H.('33) . . . . .322 Main St., Stamford, Conn., ALR
TUSAK, ERVIN A.('31) . . .115 E. 90th St., New York, N. Y., Op.

U

UHLER, S. MANN ('30) . . . . . 104 N. 8th St., Allentown, Pa., ALR
UREN, C. T.('19) . .537 City Nat'l Bank Bldg., Omaha, Nebr., ALR
*URNER, M. H. . . . .2600 Union Central Bldg., Cincinnati, O., OALR

V

*VAIL, DERRICK T.('31) . . . . . Carew Tower, Cincinnati, O., Op.
*VAIL, H. H. ('24) . . . . . Carew Tower, Cincinnati, O., ALR
*VALONE, FRANK H.('31) . . . . . 258 Genesee St., Utica, N. Y., ALR
*VAN ALYEA, O. E.('35) . . .30 N. Michigan Ave., Chicago, Ill., ALR
VAN DELLEN, A. L.('30) . . .6857 S. Halsted St., Chicago, Ill., Op.
VANDEVERE, WM. E.('32) . . . . Bassett Tower, El Paso, Tex., OALR
VAN HORN, A. F. ('30) . . .514 Central Ave., Plainfield, N. J., ALR
*VAN OSDOL, HARRY A. ('31)
828 Chamber of Commerce, Indianapolis, Ind., ALR
VAN POOLE, G. McD.('24) . . Young Bldg., Honolulu, Hawaii, OALR
VAUGHAN, G. E.('07) . . . .711 South 3rd St., Louisville, Ky., OALR
VAUGHN, E. H. . . . . Peoples Nat'l Bank Bldg., Tyler, Tex., OALR
*VEASEY, C. A., JR.('27)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
*VOIGT, C. B. ('12) . . . . . 1702 Broadway, Mattoon, Ill., OALR

*Von der Heydt, Robert ('26)
25 E. Washington St., Chicago, Ill., Op.
Voorhees, Earle W.('35).25 Market St., Poughkeepsie, N.Y., ALR
*Vorisek, Elmer A.('33).104 S. Michigan Ave., Chicago, Ill., Op.

W

Wachsberger, Alfred('31).51 W. 73rd St., NewYork,N.Y., ALR
*Wagener, Henry P.('26)...Mayo Clinic, Rochester, Minn., Op.
Wagers, Arthur J.('20).1429 Spruce St., Philadelphia, Pa., ALR
Wagner, Carl B.('12)...30 N. Michigan Ave., Chicago, Ill., OALR
*Wagner, W. A.('27)..200 Carondelet St., New Orleans, La., ALR
*Waldeck, Ed. A. ('34)
238 W. Wisconsin Ave., Milwaukee, Wis., Op.
Walker, C. B.('32).....427 W. 5th St., Los Angeles, Calif., Op.
Walker, Chas. C.('20).....Maccabee Bldg., Detroit, Mich., Op.
Walker, C. E.('18).....1732 High St., Denver, Colo., Op.
Walker, George H.('27)....172 Main St., Winona, Minn., OALR
*Walker, George W.('32)
532 T. W. Patterson Bldg., Fresno, Calif., ALR
Walker, Herbert S. ....30 N. Michigan Ave., Chicago, Ill., OALR
Walker, John R. ('32)
532 T. W. Patterson Bldg., Fresno, Calif., Op.
*Walker, O. J.('35)...Home S. & L. Bldg., Youngstown, O., ALR
Walls, Ed. G.('35).Maison Blanche Bldg., New Orleans, La., ALR
Walsh, Joseph Mark('23).....Rapid City, S. D., OALR
Ward, J. W.('30).....2607½ Lee St., Greenville, Tex., ALR
*Warner, Robert J.('29)...708 Church St., Nashville, Tenn., Op.
*Warren, William C., Jr.('28)..Doctors Bldg., Atlanta, Ga., ALR
Warwick, Harold Lynwood('28)
913 Medical Arts Bldg., Fort Worth, Texas, ALR
Watkins, Edwin Dial('26)
1412 Exchange Bldg., Memphis, Tenn., OALR
Watkins, H. R.('30)....Griesheim Bldg., Bloomington, Ill., ALR
Watson, H. deWitt('15).151 Front St., Binghamton, N. Y., OALR
Watson, James A. ..Medical Arts Bldg., Minneapolis, Minn., OALR
Watson, R. S.('23)...120 N. Michigan St., Saginaw, Mich., OALR
*Waugh, J. M.
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., OALR
*Weaver, Homer V.('32).228 Cleveland Ave. N.W.,Canton,O., ALR
Weaver, T. W.('14).....201½ N. Main St., Wichita, Kan., Op.
Weber, Lt. Comdr. Henry C.(M.C.)
U.S.S. Astoria, via Postmaster, Long Beach, Calif., OALR
Webster, F. R.('30).720 State Tower Bldg., Syracuse, N.Y., Op.
*Wechsler, A. H.('34).340 New York Ave., Brooklyn, N.Y., ALR
Weeks, Webb W.('23)....20 E. 53rd St., New York, N. Y., Op.
*Weeth, C. R.('30).....37 Remsen St., Brooklyn, N. Y., ALR
Weidlein, Ivan F.('30).10515 Carnegie Ave., Cleveland, O., ALR
*Weih, E. P. ('20).....217 Fifth Ave. South, Clinton, Ia., OALR
Weil, A. I. .....3503 Prytania St., New Orleans, La., ALR

WEINBERGER, NELSON S.('28).Robt. Packer Hosp., Sayre,Pa., OALR
WEINSTEIN, A. J.('35)....244 W. 74th St., New York, N. Y., ALR
WEINSTEIN, M. A.('27)...1309 Spruce St., Philadelphia, Pa., ALR
WEISS, J. ALLAN('32)....104 S. Michigan Ave., Chicago, Ill., ALR
WEISSER, E. A. .....806 May Bldg., Pittsburgh, Pa., Op.
*WELCH, ROBERT S. G.('22)..86 State Circle, Annapolis, Md., OALR
WELDEN, WM. A.('33).....Glasgow, Ky., Op.
WELLS, GEORGE S.('17).8 E. Arrellaga, Santa Barbara, Calif., ALR
WELSH, THOMAS F.('34).Boston Bldg., Salt Lake City, Utah, OALR
WELTY, C. F.('26).....210 Post St., San Francisco, Calif., ALR
*WENCKE, CARL G....Battle Creek Sanit., Battle Creek, Mich., ALR
*WENDEL, J. S.('23).744 David Whitney Bldg., Detroit, Mich., ALR
WENNER, W. T.('30)....St. Mary's Bldg., St. Cloud, Minn., Op.
WESCOTT, VIRGIL('35)....30 N. Michigan Ave., Chicago, Ill., Op.
WESSELS, ANDREW B.('29)
Nix Professional Bldg., San Antonio, Tex., ALR
*WESTERVELT, H. O. ('32)
239 Pipestone St., Benton Harbor, Mich., ALR
WESTLAKE, SAMUEL B..Beaumont Med. Bldg., St. Louis, Mo., ALR
WETHERBEE, CHARLES('26)..Booth Bldg., Twin Falls, Idaho, OALR
*WETZEL, JOHN O.('30)..124 W. Allegan St., Lansing, Mich., Op.
WEXLER, DAVID('30).....179 E. 79th St., New York, N. Y., Op.
WHALEN, EDWARD JOS.('28)..750 Main St., Hartford, Conn., ALR
*WHEELER, J. M.('20)...635 W. 165th St., New York, N. Y., Op.
WHEELER, MAYNARD C.('35).30 W. 59th St., New York,N.Y., Op.
*WHEELER, MERRITT W. ('23)
1027 Lowry Bldg., St. Paul, Minn., OALR
WHELAN, G. L. ('25)...2100 Walnut St., Philadelphia, Pa., ALR
WHELAN, WM. F.('34)..2015 Walnut St., Philadelphia, Pa., ALR
*WHISNANT, A.M.('13).517 Professional Bldg.,Charlotte,N.C., OALR
WHITAKER, J.('17).Hume-Mansur Bldg., Indianapolis, Ind., OALR
WHITE, CHAS. P.('22)
507 Citizens Bk. Bldg., Wilmington, Del., OALR
*WHITE, JAMES W.('34).....15 Park Ave., New York, N. Y., Op.
WHITE, VIVIAN P.('31)
Ezra Thompson Bldg., Salt Lake City, Utah, ALR
WHITE, WM. BEVERLY('28)..Gurley Bldg., Stamford, Conn., OALR
WHITNEY, E. L.('30)..Henry Ford Hospital, Detroit, Mich., ALR
*WIBLE, LEROY E.('35).....Empire Bldg., Pittsburgh, Pa., ALR
*WICKLIFFE, THOMPSON F.('31)..Tweedy Bldg., Jasper, Ala., ALR
*WIENER, MEYER ('17)...900 Carleton Bldg., St. Louis., Mo., Op.
WIGGIN, WILLIAM I. ('29)...202 Central St., Lowell, Mass., ALR
*WILKERSON, WM. W., JR. ('32)
Bennie-Dillon Bldg., Nashville, Tenn., ALR
WILKINS, SAMUEL H. ('34)
270 Commonwealth Ave., Boston, Mass., Op.
*WILKINSON, A. P.('30).....Fisher Bldg., Detroit, Mich., ALR
*WILKINSON, OSCAR('30).1408 L St., N.W., Washington,D.C., OALR

*Wilkinson, Richard W.('34)
1408 L St. N.W., Washington, D. C., ALR
*Williams, Horace J.('22).5908 Greene St., Germantown, Pa., ALR
*Williams, M. M.('27)...Medical Arts Bldg., Scranton, Pa., Op.
*Williams, Walton H.('21)..Castell Bldg., Middletown, O., OALR
Willson, Wesley W.('28).2204 Eaton Tower, Detroit,Mich., OALR
Wilson, Frank E.('33)..302 State St., New London, Conn., ALR
*Wilson, Robert G. ('31)..433 E. Main St., Batavia, N. Y., ALR
Wincor, Henry G. ('31)
1882 Grand Concourse, New York, N. Y., Op.
*Windham, Robert Edward ('28)
234 W. Beauregard Blvd., San Angelo, Texas, OALR
*Winger, Ira B.('27).....225 Michigan St., Toledo, O., OALR
Winkler, Herman('34)..224 Thayer St., Providence, R. I., ALR
*Winter, George E.('22).....Rogers Bldg., Jackson, Mich., OALR
*Winter, John A. ('23).....205 W. 2nd St., Duluth, Minn., OALR
Wise, Ralph C.('14).....News Bldg., Mansfield, O., OALR
Wolcott, Chas. C.('31).....27 Ludlow St., Yonkers, N. Y., ALR
Wold, K. C.('24)
1027 Lowry Med. Arts Bldg., St. Paul, Minn., OALR
*Wolfe, Claude T. ('17)....Heyburn Bldg., Louisville, Ky., OALR
Wolfe, Otis R.('19).415 Masonic Temple, Marshalltown, Ia., Op.
Womack, David R.('33)
Maison Blanche Bldg., New Orleans, La., ALR
Wood, Earl LeRoy('28)..172 Roseville Ave., Newark, N. J., ALR
Wood, Ed. C.('35).....405 Park Ave., New York, N. Y., Op.
also 80 Maple Ave., White Plains, N. Y.
Wood, V. Visscher('28)
201 Beaumont Medical Bldg., St. Louis, Mo., ALR
Woodman, Degraaf('33).....156 E. 37th St., ALR
Woodruff, Frederick E..Metropolitan Bldg., St. Louis, Mo., Op.
*Woodruff, Geo. H.('32).....500 N. Ottawa St., Joliet, Ill., ALR
*Woodruff, H. W. .....500 N. Ottawa St., Joliet, Ill., OALR
Woodruff, Walter Stuart ('27)
421 Huguenot St., New Rochelle, N. Y., ALR
Woods, R. H. .....Neustadt Bldg., La Salle, Ill., Op.
Woodson, Burbank P.('28).Woodson Hosp., Temple, Tex., Op.
*Woodson, Warren B.('33).5th St. & Ave. G, Temple, Tex., Op.
*Woodward, F. D. ('29).....Box 1685, University, Va., ALR
Woodworth, Wm. P.('27).2994 E.Grand Blvd.,Detroit,Mich., OALR
*Wright, J. Wm. ....Hume-Mansur Bldg., Indianapolis, Ind., ALR
Wright, Robt. P.('32).1414 Drummond St., Montreal, Can., ALR
Wright, Walter W.('26)..Med. Arts Bldg., Toronto, Can., Op.
*Wurtz, W. J. M.('13)...333 Linwood Ave., Buffalo, N. Y., ALR

Y

Yates, Earl C.('31).....190 N. Upper St., Lexington, Ky., ALR
Yazel, Herman E. .....733 Rialto Bldg., Kansas City, Mo., OALR

DE YOANNA, A. A.('30)..111 Pierrepont St., Brooklyn, N.Y., ALR
*YOST, WALTER M.('28)..Peoples Bank Bldg., Rochester, Pa., Op.
Also Westinghouse Bldg., Pittsburgh, Pa.
*YOUNG, CHARLES A.('26).Medical Arts Bldg., Roanoke, Va., Op.
YOUNG, CLARENCE M.('35)...305 S. H St., Aberdeen, Wash., ALR

Z

*ZEHNDER, A. CHAS.('19)...188 Roseville Ave., Newark, N. J., Op.
ZEMER, S. G.('17).....Stuart Bldg., Lincoln, Nebr., OALR
ZENTMAYER, WM. .....1930 Chestnut St., Philadelphia, Pa., Op.
ZINKHAN, A. M.('20).1801 Eye St. N.W., Washington, D. C., ALR
*ZUBAK, MATTHEW FRANCIS ('28)
Citizens Mutual Trust Bldg., Wheeling, W. Va., OALR

Geographical List of Members

ALABAMA

BIRMINGHAM

KINCANNON, LEROY T. ..... 1927 First Ave. N., OALR
MAUMENEE, A. E. ..... 711 Empire Bldg., OALR
POPE, ERNEST C. ..... The Woodward Bldg., ALR
RUCKER, E. W., JR. ..... 926 Woodward Bldg., ALR
STILES, PORTER ..... Medical Arts Bldg., ALR

DOTHAN

KEYTON, JOHN A. ..... 108½ E. Main St., ALR

JASPER

WICKLIFFE, T. F. ..... Tweedy Bldg., ALR

MOBILE

PERDUE, JAMES D. ..... Merchants Nat'l Bank Bldg., Op.
SANDERS, JOHN GILLIS ..... Merchants Nat'l Bank Bldg., ALR

MONTGOMERY

MERTINS, PAUL S. ..... 1115 Bell Bldg., OALR
THIGPEN, C. A. ..... 401 S. Court St., OALR

SELMA

KIRKPATRICK, SAMUEL ..... 902 Alabama Ave., OALR

TUSCALOOSA

SEARCY, H. B. ..... 2501 Sixth St., OALR

ARIZONA

MESA

MCCARTNEY, OLIVER P. ..... Valley Bank Bldg., OALR

PHOENIX

MCLOONE, JOHN J. ..... Professional Bldg., OALR
MELTON, B. L., JR. ..... 15 E. Monroe St., Op.
MOSS, H. E. ..... Professional Bldg., OALR
ROBB, MAYO ..... Professional Bldg., ALR
SCHWARTZ, WILLIAM A. ..... Professional Bldg., OALR

PRESCOTT

SWEETMAN, C. R. K. ..... Masonic Temple, ALR

TUSCON

STOKES, HARRY B. ..... 130 S. Scott St., ALR

ARKANSAS

FORT SMITH

BUCKLEY, J. H. .....1st National Bank Bldg., OALR
DORENTE, J. H. .....820 S. 25th St., OALR
MOULTON, EVERETT C. .....Merchants National Bank Bldg., OALR
MOULTON, HERBERT .....Merchants National Bank Bldg., OALR

HOT SPRINGS

KING, O. H. .....Medical Arts Bldg., OALR

LITTLE ROCK

CALDWELL, ROBERT .....831 New Donaghey Bldg., OALR
MAHONEY, PAUL LEO .....Donaghey Bldg., ALR
MOORE, ROBERT B. .....2000 Main St., ALR
VINSONHALER, F. .....Urquhart Bldg., OALR

PINE BLUFF

PAYNE, VIRGIL LEROY .....Simmons Nat'l Bldg., ALR
SCALES, J. W. .....204½ Main St., OALR

ROGERS

KOOBS, HERMAN J. G. .....117½ W. Walnut St., OALR

CALIFORNIA

ALHAMBRA

BOBB, E. V. .....2 Garfield Ave., OALR

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THOMSEN, VON COLDITZ G. .....25 E. Washington St., OALR
TINDAL, JOHN M. .....4738 W. Washington Blvd., OALR
TIVNEN, RICHARD J. .....30 N. Michigan Blvd., OALR
VAN ALYEA, O. E. .....30 N. Michigan Ave., ALR
VAN DELLEN, A. L. .....6857 S. Halsted St., Op.
VON DER HEYDT, ROBERT .....25 E. Washington St., Op.
VORISEK, ELMER A. .....104 S. Michigan Ave., Op.
WAGNER, CARL B. .....30 N. Michigan Ave., OALR
WALKER, HERBERT S. .....30 N. Michigan Ave., OALR
WEISS, J. ALLAN .....104 S. Michigan Ave., ALR
WESCOTT, VIRGIL .....30 N. Michigan Ave., Op.
WOOD, CASEY A. .....7 W. Madison St., Op.

CHICAGO HEIGHTS

GORRELL, T. J. H. .....16th and Vincennes Ave., OALR

DANVILLE

BALDWIN, H. E. .....Temple Bldg., ALR
CARMODY, THOMAS J. .....408 The Temple Bldg., OALR

DIXON

MCNICHOLS, W. A. .....101 W. 1st St., OALR

EAST ST. LOUIS

HANSON, WM. LUDWIG .....707 Spivey Bldg., ALR
SPITZE, E. C. .....Murphy Bldg., Op.

EVANSTON

DODD, OSCAR .....636 Church St., Op.
GALLOWAY, T. C. .....636 Church St., ALR
GREENWOOD, G. J. .....636 Church St., ALR
LAWSON, L. J. .....636 Church St., ALR
SOPER, GAIL RANEY .....636 Church St., Op.
SWAN, C. J. .....636 Church St., OALR

FREEPORT

CLARK, J. SHELDON .....509 State Bank Bldg., OALR

GALESBURG

MATHENY, R. C. .....306 E. Main St., OALR

GREAT LAKES

STONE, LESLIE O., LT. COMDR. (M.C.) U. S. Navy
U. S. Naval Hospital, ALR

JACKSONVILLE

ADAMS, A. L. .....Professional Bldg., OALR

JOLIET

WOODRUFF, GEO. H. .....500 N. Ottawa St., ALR
WOODRUFF, H. W. .....500 N. Ottawa St., OALR

KANKAKEE

GEIGER, C. W. .....Arcade Bldg., OALR

LA SALLE

WOODS, R. H. .....Neustadt Bldg., Op.

MACOMB

DUNTLEY, G. S. .....133 S. Randolph St., OALR

MATTOON

VOIGT, C. B. .....1702 Broadway, OALR

MOLINE

FLATLEY, T. J. .....Reliance Bldg., OALR

OAK PARK

DVORAK-THEOBALD, GEORGIANA .....715 Lake St., Op.
SPIESMAN, IRWIN G. .....715 Lake St., ALR

OTTAWA

GUIBOR, GEO. P. .....Central Life Bldg., Op.

PEKIN

GLASFORD, S. T. .....333 Court St., OALR

PEORIA

BLICKENSTAFF, A. J. ..... Alliance Life Bldg., OALR
BROBST, C. H. ..... Central National Bank Bldg., OALR
DUANE, J. F. ..... Jefferson Bldg., OALR
FLOYD, T. W. ..... 811 Peoria Life Bldg., OALR
THOMAS, C. D. ..... Central National Bank Bldg., OALR
WILLIAMS, W. C. ..... Jefferson Bldg., OALR

PONTIAC

MIDDLETON, A. B. ..... Sterry Blk., OALR

QUINCY

GABRIEL, C. K. ..... Illinois State Bank Bldg., ALR
MERCER, RAY ..... W. C. U. Bldg., OALR
STEVENSON, WALTER ..... W. C. U. Bldg., OALR

ROCKFORD

BLOMBERG, T. E. ..... 501 Seventh St., ALR
FRINGER, W. R. ..... William Brown Bldg., Op.

SPRINGFIELD

DEAL, JOHN FRANCIS ..... First Nat'l Bank Bldg., OALR
HAGLER, E. E. ..... 401 East Capitol Ave., OALR

STERLING

BRODRICK, FRANK W. ..... 301 First Ave., OALR

STREATOR

LESTER, HARRY S. ..... 420 Murray Bldg., OALR

INDIANA

CRAWFORDSVILLE

LINGEMAN, B. N. ..... Ben Hur Bldg., ALR

EVANSVILLE

KNAPP, B. J. ..... Citizens' Bank Bldg., OALR
RAVDIN, B. D. ..... Central Union Bank Bldg., OALR
RAVDIN, M. ..... Central Union Bank Bldg., OALR
STANTON, HARMON LAM. ..... Central Union Bank Bldg., OALR

FORT WAYNE

BULSON, EUGENE LORING ..... 406 W. Berry St., OALR
GESSLER, WM. F. ..... Medical Arts Bldg., Op.
MCBRIDE, W. O. ..... 2902 S. Fairfield Ave., OALR
O'ROURKE, CARROLL ..... 347 W. Berry St., ALR

HAMMOND

KUHN, HEDWIG S. ..... 5231 Hohman Ave., Op.
KUHN, HUGH A. ..... 5231 Hohman Ave., ALR
LLOYD, A. W. ..... 804 Lloyd Bldg., OALR
SHANKLIN, E. M. ..... 575 Hohman St., OALR

INDIANAPOLIS

ADAMS, D. S. ..... Hume-Mansur Bldg., OALR
ARONSON, SIDNEY S. ..... Hume-Mansur Bldg., ALR
BERRY, D. F. ..... Bankers Trust Bldg., ALR
CHAPPELL, R. S. ..... Hume-Mansur Bldg., ALR
CLARK, CECIL P. ..... 24 E. Ohio St., Op.
CLEVENGER, W. F. ..... Hume-Mansur Bldg., ALR
CRAFT, KENNETH L. ..... Hume-Mansur Bldg., ALR
DEARMIN, ROBERT M. ..... Hume-Mansur Bldg., ALR
ELLIS, BERT ED. ..... Hume-Mansur Bldg., ALR
GARRETT, JOHN D. ..... Bar Association Bldg., ALR
HUGHES, W. F. ..... 401 Hume-Mansur Bldg., Op.
KEARBY, D. O. ..... Hume-Mansur Bldg., ALR
LARKIN, BERNARD J. ..... 23 E. Ohio St., Op.
LEASURE, JOHN K. ..... Hume-Mansur Bldg., ALR
LINGEMAN, EDWARD LAUREL ..... 406 Pennway Bldg., ALR
LITTELL, J. J. ..... Hume-Mansur Bldg., ALR
MCCASKEY, C. H. ..... Guaranty Bldg., ALR
MASTERS, ROBERT J. ..... 23 E. Ohio St., Op.
MOLT, WM. F. ..... 529 Bankers Trust Bldg., ALR
MUELLER, LILLIAN BARBARA ..... Continental Bank Bldg., ALR
NEWCOMB, JOHN R. ..... 410 Hume-Mansur Bldg., Op.
OVERMAN, F. V. ..... Hume-Mansur Bldg., ALR
PFAFFLIN, C. A. ..... 445 Bankers Trust Bldg., OALR
ROW, D. HAMILTON ..... 23 E. Ohio St., Op.
ROW, GEORGE S. ..... 906 Hume-Mansur Bldg., Op.
RUTHERFORD, C. W. ..... Hume-Mansur Bldg., Op.
TOMLIN, W. S. ..... Hume-Mansur Bldg., ALR
VAN OSDOL, HARRY A. ..... Chamber of Commerce Bldg., ALR
WHITAKER, J. ..... Hume-Mansur Bldg., OALR
WRIGHT, J. WM. ..... Hume-Mansur Bldg., ALR

KOKOMO

ADAMS, CHAS. JOSEPH ..... Armstrong-Landon Bldg., OALR

LOGANSPORT

EGAN, BURTON WRIGHT ..... 408 North St., OALR
HICKMAN, WARREN R. ..... 211 Third St., ALR
HOLMES, WILL W. ..... Masonic Temple, ALR

MARION

BRAUNLIN, WM. H. ..... Marion Nat'l Bank Bldg., OALR

MICHIGAN CITY

KRIEGER, G. M. ..... 701 Washington St., OALR

RICHMOND

HOLLAND, EMORY ED. ..... 51 S. 8th St., Op.

SOUTH BEND

BOONE, J. C. ..... Sherland Bldg., ALR
BOYD-SNEE, HARRY ..... 716 J. M. S. Bldg., ALR
CASSADY, J. VERNAL ..... 654 Associates Bldg., OALR
EDWARDS, SLOCOMB R. ..... Christman Bldg., OALR
LENT, E. J. ..... 122 N. Lafayette Blvd., OALR
RIGLEY, ED. L. ..... Sherland Bldg., ALR
SAVERY, C. E. ..... 206 Sherland Bldg., OALR

TERRE HAUTE

ALLEN, ORRIS T. ..... 422 Rose Dispensary Bldg., OALR
GILLUM, J. R. ..... 221 S. 6th St., OALR
MILLER, WM. H. ..... Tribune Bldg., ALR
RHEIN, ALFRED E. ..... Tribune Bldg., ALR
STEWART, WALTER E. ..... Tribune Bldg., OALR

UNION CITY

RUBY, F. McK. ..... 235 W. Pearl St., OALR

WARSAW

HOWARD, C. NORMAN ..... Warsaw Clinic, OALR

IOWA

BURLINGTON

LA FORCE, E. F. ..... Iowa State Bank Bldg., OALR

CEDAR RAPIDS

BAILEY, FRED W. ..... Merchants Nat'l Bank Bldg., OALR
IVINS, H. M. ..... 815 Merchants Nat'l Bank Bldg., OALR
FOSTER, WAYNE J. ..... Higley Bldg., ALR

CLINTON

FOSTER, WARREN H. ..... 217 Fifth Ave., S., ALR
WEIH, E. P. ..... 217 Fifth Ave. South, OALR

COUNCIL BLUFFS

DEAN, F. W. ..... 532 First Ave., OALR
HOWARD, LLOYD G. ..... Council Bluffs Clinic, OALR
MAIDEN, SYDNER D. ..... 403 Bennett Bldg., OALR

CRESTON

CHILDS, H. A. ..... Eagle Blk., OALR

DAVENPORT

HANDS, S. GILLOTT ..... 201 West Second St., ALR
HARKNESS, GORDON F. ..... Putnam Bldg., OALR
HOFMANN, W. P. ..... 707 Kahl Bldg., OALR
LAMB, HARRY HOWE ..... Davenport Bank Bldg., ALR

DES MOINES

JONES, CECIL C. ..... Equitable Bldg., OALR
LYNCH, ROBERT J. ..... Equitable Bldg., ALR
MUMMA, CLAUDE S. ..... Hotel Fort Des Moines, ALR
PEARSON, WM. W. ..... 417 Bankers Trust Bldg., OALR

DUBUQUE

THOMPSON, HOWARD E. ..... 1100 Main St., OALR

FORT DODGE

CHASE, S. B. ..... Carver Bldg., OALR

FORT MADISON

REIMERS, R. S. ..... 613 Eighth St., ALR

IOWA CITY

ALBRIGHT, GEORGE C. ..... Johnson County Bank Bldg., ALR
BOILER, W. F. ..... Dey Bldg., OALR
LIERLE, DEAN MCA. ..... University Hospital, ALR
MOORE, PAUL, JR. ..... University Hospital, ALR
O'BRIEN, C. S. ..... University Hospital, Op.
POTTER, JACOB J. ..... University Hospital, ALR
THYGESON, PHILLIPS ..... Dept. of Ophth., Univ. Hosp., Op.

KEOKUK

LAPSLEY, ROBERT M. ..... Masonic Temple, OALR

LE MARS

JOYNT, M. J. ..... Royal Bldg., ALR

MARSHALLTOWN

COBB, EDWIN ..... Masonic Temple Bldg., OALR
FRENCH, R. F. ..... Masonic Temple Bldg., OALR
WOLFE, OTIS R. ..... 415 Masonic Temple, Op.

MASON CITY

FALLOWS, HOWARD D. ..... Foresters Bldg., OALR
O'BRIEN, STEVE A. ..... First Nat'l Bank Bldg., OALR

MUSCATINE

GOAD, R. R. ..... Laurel Bldg., Op.

NEWTON

ENGLE, H. P. ..... 212 West First Ave., OALR

OTTUMWA

TAYLOR, CHARLES B. ..... 118 E. Second St., ALR

SHENANDOAH

MALOY, WAYLAND H. .....608½ W. Sheridan Ave., Op.

SIOUX CITY

DECKER, J. C. .....Frances Bldg., OALR
GITTINS, T. R. .....401 Davidson Bldg., OALR
NAFTZGER, J. B. .....Davidson Bldg., OALR
REEDER, J. E. .....Davidson Bldg., OALR
SALLANDER, F. W. .....New Orpheum Bldg., OALR
SECOY, F. L. .....301 Davidson Bldg., OALR
TRIPP, LEROY R. .....Security Bldg., OALR

WATERLOO

BENDER, H. A. .....Black Bldg., ALR
JOYNT, A. J. .....Black Bldg., OALR
REULING, F. H. .....Black Bldg., OALR
RUSSELL, R. E. .....606 Commercial St., OALR

WAVERLY

ROBINSON, R. E. .....205½ E. Bremer Ave., OALR

KANSAS

ATCHISON

BRIBACH, EUGENE J. .....Simpson Bldg., OALR

COFFEYVILLE

CHADWICK, IRA B. .....Terminal Bldg., Op.

CONCORDIA

ROBERTSON, EDW. N. .....New Caldwell Bldg., OALR

ELDORADO

SHULTS, E. J. G. .....Haines Bldg., OALR

EMPORIA

GRANGER, W. B. .....Gazette Bldg., OALR
TRIMBLE, CLARENCE S. .....Gazette Bldg., OALR

HUTCHINSON

GREEVER, BOYD L. .....100 No. Main St., OALR
QUIRING, WALTER O. .....100 First West St., ALR
SCALES, H. L. .....Rorabaugh Wiley Bldg., OALR
SCHRANT, J. H. .....14 West First St., OALR

KANSAS CITY

LIDIKAY, C. J. .....903 N. Seventh St., OALR
MULLEN, CLIFFORD J. .....Brotherhood Bldg., Op.
SPAKE, LAVERNE B. .....Brotherhood Bldg., ALR

LAWRENCE

POWELL, LYLE S. ..... 833½ Mass. St., OALR

TOPEKA

ALKIRE, H. L. ..... 614 Kansas Ave., OALR
ALLEN, GEORGE V. ..... 835 Kansas Ave., OALR
ALLEN, G. HENRY ..... Mills Bldg., OALR
DYER, BENJAMIN ..... Santa Fe Hospital, ALR
REED, WM. W. ..... Mills Bldg., ALR

WICHITA

BROWNELL, MORTON EMMONS ..... 105 N. Main St., Op.
CARTER, E. D. ..... Central Bldg., ALR
CHENEY, JAMES W. ..... 702 Schweiter Bldg., OALR
GILLETT, WILBUR G. ..... 201½ N. Main St., Op.
GSELL, J. F. ..... 911 Beacon Bldg., OALR
MAGGARD, D. I. ..... 715 Orpheum Bldg., OALR
MARSHALL, HAL E. ..... 227 E. Douglas St., ALR
SEYDELL, E. M. ..... 1030 First Nat'l Bank Bldg., ALR
TIPPIN, ERNEST E. ..... 601 First Nat'l Bank Bldg., ALR
WEAVER, T. W. ..... 201½ N. Main St., Op.

WINFIELD

RALLS, C. T. ..... First National Bank Bldg., OALR

KENTUCKY

BEREA

COWLEY, ROBERT HENRY ..... , OALR

COVINGTON

BLEDSOE, R. W. ..... 1005 Madison Ave., OALR

GLASGOW

WELDON, WILLIAM A. ..... Op.

HARLAN

GUNN, MILUS LYDELL ..... Smith Bldg., ALR

LEXINGTON

ARMSTRONG, R. M. ..... First National Bank Bldg., OALR
DEWEESE, CLARENCE ..... 201 West Second St., OALR
MARKS, SAMUEL BLACKBURN ..... Security Trust Bldg., ALR
YATES, EARL C. ..... 190 N. Upper St., ALR

LOUISVILLE

BASS, A. L. ..... Heyburn Bldg., OALR
DABNEY, S. G. ..... Starks Bldg., OALR
DEAN, WALTER ..... 300 Francis Bldg., OALR
DULANEY, OCTAVIUS ..... 811 Brown Bldg., ALR

HALL, G. C. ..... 705 Brown Bldg., OALR
HEITGER, J. D. ..... Heyburn Bldg., OALR
LEGGETT, ALBERT E. ..... 614 Breslin Bldg., Op.
PFINGST, A. O. ..... Heyburn Bldg., Op.
SHAFER, J. J. ..... Starks Bldg., OALR
STERN, MILTON J. ..... First Nat'l Bank Bldg., Op.
TOWNES, C. DWIGHT ..... Heyburn Bldg., Op.
VAUGHAN, G. E. ..... 711 S. Third St., OALR
WOLFE, CLAUDE T. ..... Heyburn Bldg., OALR

PADUCAH

PACE, ERRETT ..... City National Bank Bldg., ALR
REYNOLDS, H. G. ..... Citizens Savings Bank Bldg., OALR

RICHMOND

DUNN, MURISON ..... OALR

LOUISIANA

LAKE CHARLES

MOSS, OLIN W. ..... Weber Bldg., ALR

NEW ORLEANS

ALEXANDER, LUCIAN W. ..... 1230 Maison Blanche Bldg., ALR
BAHN, CHAS. A. ..... Pere Marquette Bldg., Op.
BLUM, HENRY N. ..... American Bank Bldg., Op.
BREWSTER, H. F. ..... Union Indemnity Bldg., ALR
BROWN, M. EARLE ..... 921 Canal St., Op.
BUFFINGTON, WILEY R. ..... Hibernia Bank Bldg., Op.
CLARK, WM. B. ..... American Bank Bldg., Op.
FUCHS, VALENTINE HENRY ..... American Bank Bldg., ALR
GOOCH, JOHN B. ..... Canal Bank Bldg., ALR
GRANBERRY, CARL ..... Maison Blanche Bldg., ALR
HASPEL, M. DAVID ..... Maison Blanche Bldg., OALR
HUME, JOHN R. ..... Maison Blanche Bldg., ALR
LANDFRIED, C. J. ..... 607 Baronne Bldg., ALR
LEJEUNE, F. E. ..... 632 Maison Blanche Bldg., ALR
SMITH, VICTOR C. ..... American Bank Bldg., Op.
TAQUINO, GEORGE J. ..... 208 Audubon Bldg., ALR
WAGNER, W. A. ..... 200 Carondelet St., ALR
WALLS, ED. G. ..... Maison Blanche Bldg., ALR
WEIL, A. I. ..... 3503 Prytania St., ALR
WOMACK, DAVID R. ..... Maison Blanche Bldg., ALR

SHREVEPORT

LARUE, C. L. ..... Highland Clinic, OALR
SCALES, J. L. ..... Medical Arts Bldg., OALR

MAINE

AUGUSTA

TURNER, OLIVER W. ..... Masonic Temple, OALR

BANGOR

JOHNSON, JOHN L. ..... 33 Grove St., ALR

BATH

KERSHNER, W. E. ..... 117 Front St., ALR

LEWISTON

CHAFFERS, WM. H. ..... 190 Bates St., ALR

PORTLAND

BEACH, S. JUDD ..... 704 Congress St., Op.
CUMMINGS, GEORGE O. ..... 47 Deering St., ALR
FISHER, STANWOOD E. ..... 723 Congress St., ALR
GILBERT, FRANK Y. ..... 148 Park St., OALR
GORDON, CHAS. H. ..... 46 Deering St., ALR
HASKELL, ALFRED W. ..... 142 High St., Op.
HOLT, E. E., JR. ..... 723 Congress St., Op.
JOHNSON, HENRY P. ..... 32 Deering St., ALR
MCADAMS, WM. R. ..... 704 Congress St., Op.
O'SULLIVAN, T. J. ..... 10 Pine St., ALR
SPALDING, J. A. ..... 627 Congress St., Op.A

ROCKLAND

ELLINGWOOD, WM. A. ..... 420 Main St., ALR

WATERVILLE

HILL, FREDERICK T. ..... Professional Bldg., ALR
HILL, HOWARD F. ..... Professional Bldg., Op.

MARYLAND

ANNAPOLIS

WELCH, ROBERT S. G. ..... 86 State Circle, OALR

BALTIMORE

BORDLEY, JAMES, JR. ..... 300 N. Charles St., OALR
FRIEDENWALD, HARRY ..... 1212 Eutaw Pl., Op.A
FRIEDENWALD, J. S. ..... 1212 Eutaw Pl., Op.
GOLDBACK, L. J. ..... 1012 N. Charles St., Op.
KEMLER, JOS. I. ..... 1908 Eutaw Pl., OALR
LOOPER, EDWARD ANDERSON ..... 104 W. Madison St., OALR
MACLEAN, ANGUS L. ..... 1201 N. Calvert St., Op.
PETERMAN, H. E. ..... 512 Cathedral St., OALR
TARUN, WILLIAM ..... 104 W. Madison St., Op.

CUMBERLAND

JONES, E. L. ..... First National Bank Bldg., OALR
ROBINSON, H. T. ..... Medical Bldg., OALR
SHARRETT, GEORGE O. ..... 119 Bedford St., OALR

MASSACHUSETTS

BEVERLY

ODENEAL, THOMAS HELM .....21 Broadway, OALR

BOSTON

BRYANT, ALICE GERTRUDE .....405 Marlborough St., ALR
CAHILL, HARRY P. .....520 Commonwealth Ave., ALR
CASTEN, VIRGIL G. .....520 Commonwealth Ave., Op.
CHENERY, W. E. .....377 Commonwealth Ave., ALR
CLINE, SAMUEL .....353 Commonwealth Ave., ALR
COWAN, CLYDE R. .....Hotel Buckminster, ALR
DUNPHY, ED. B. .....101 Bay State Road, Op.
EVANS, MAURICE GEO. .....311 Commonwealth Ave., ALR
FREEDMAN, L. M. .....475 Commonwealth Ave., ALR
GARLAND, FREDERICK E. .....483 Beacon St., LR
GOODALL, EDWIN BAKER .....101 Bay State Road, Op.
GREENWOOD, ALLEN .....82 Commonwealth Ave., Op.
GUNDERSEN, TRYGVE .....5 Bay State Road, Op.
HALEY, WM. THOS. .....531 Beacon St., ALR
HARTSHORNE, ISAAC .....22 Commonwealth Ave., Op.
HOOVER, WALTER BOYD .....605 Commonwealth Ave., ALR
HURLEY, E. D. .....419 Boylston St., Op.
INGLIS, HARRY J. .....43 Bay State Road, ALR
JOHNS, JUANITA PEARL .....313 Commonwealth Ave., Op.
KAZANJIAN, V. H. .....475 Commonwealth Ave., LR
KELLEY, VINCENT J. .....520 Commonwealth Ave., ALR
KLEINERT, MARGARET NOYES .....82 Commonwealth Ave., ALR
KNOWLTON, CHARLES DAVISON .....520 Commonwealth Ave., ALR
LANCASTER, W. B. .....520 Commonwealth Ave., Op.
LEWIS, J. P. .....479 Commonwealth Ave., ALR
LOWELL, W. HOLBROOK .....82 Commonwealth Ave., Op.
LURIE, MOSES H. .....483 Beacon St., ALR
MELTZER, PHILIP E. .....20 Charlesgate West, ALR
MILLS, CHESTER R. .....520 Commonwealth Ave., ALR
MINITER, FRANCIS G. .....419 Boylston St., ALR
MOSHER, HARRIS P. .....828 Beacon St., ALR
MYSEL, PHILIP .....311 Commonwealth Ave., ALR
POIRIER, GEORGE H. .....60 Bay State Road, ALR
PORTER, CHAS. T. .....520 Commonwealth Ave., ALR
QUINCY, J. E. .....270 Commonwealth Ave., ALR
REGAN, JAS J. .....520 Commonwealth Ave., Op.
RICE, G. B. .....99 Bay State Road, ALR
RICHARDS, L. G. .....319 Longwood Ave., ALR
ROWLAND, W. D. .....84 Commonwealth Ave., Op.
SACHS, BENJAMIN .....483 Beacon St., Op.
SCHALL, LEROY A. .....270 Commonwealth Ave., ALR
SKIRBALL, J. J. .....353 Commonwealth Ave., Op.
TERRY, THEODORE L. .....128 Newbury St., Op.
THOMPSON, PETER H. .....308 Commonwealth Ave., Op.

TINGLEY, LOUISA PAINE .....9 Massachusetts Ave., Op.
TOBEY, GEORGE L., JR. .....270 Commonwealth Ave., ALR
WELLS, DAVID W. .....Hotel Westminster, Op.
WILKINS, SAMUEL HENRY .....270 Commonwealth Ave., Op.

CHICOPEE

GALLAGHER, JOHN H. C. .....105 Center St., ALR

CLINTON

MONAHAN, J. A. .....181 Chestnut St., OALR

FALL RIVER

BEST, LYNWOOD K. .....151 Rock St., Op.
RYDER, D. R. .....151 Rock St., ALR

FRAMINGHAM

TOMB, EVERETT H. .....46 Lexington St., ALR

GARDNER

BONE, HERMAN D. .....19 Pleasant St., OALR

GLOUCESTER

DEXTER, MILTON A. .....Cor. Pleasant and Prospect Sts., ALR
ODENEAL, THOMAS H. .....40 Middle St., OALR

GREENFIELD

MILLETT, FRANK A. .....58 Federal St., OALR

HOLYOKE

BURKE, JOHN R. .....207 Elm St., OALR

LAWRENCE

CONLON, F. A. .....301 Essex St., OALR

LOWELL

BERMAN, PHILIP G. .....174 Central St., Op.
WIGGIN, WM. IRVING .....202 Central St., ALR

MALDEN

HAIRE, PAUL G. .....6 Pleasant St., Op.

LYNN

JONES, E. W. .....44 Atlanta St., Op. A
O'REILLY, W. F. .....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F. .....232 Walnut St., OALR

NORTHAMPTON

COLLINS, J. D. ..... 187 Main St., ALR
DOW, FRANK E. ..... 4 West St., Op.

PALMER

MOORE, G. A. ..... 483 Main St., OALR

PITTSFIELD

THOMPSON, G. H. ..... 74 North St., OALR

SALEM

HENNESSEY, W. W. ..... 333 Essex St., OALR

SPRINGFIELD

BOSS, EUGENE G. ..... 1537 Main St., Op.
BYRNES, H. F. ..... 67 Chestnut St., OALR
CARLETON, RALPH ..... 10 Temple St., Op.
GAUDREAU, HONORE E. ..... 293 Bridge St., Op.
GOODELL, WILLIAM ..... 121 Chestnut St., ALR
IRWIN, VINCENT J., JR. ..... 1634 Main St., OALR
IRWIN, V. J., SR. ..... 1634 Main St. ,OALR
SEGAL, SAMUEL, JR. ..... 1562 Main St., ALR

TAUNTON

BAKER, HARRY B. ..... 59 Main St., OALR

WALTHAM

LEWIS, J. P. ..... 681 Main St., ALR

WORCESTER

BERRY, GORDON ..... 36 Pleasant St., ALR
CROISSANT, CHAS. A. ..... 390 Main St., ALR
CROSS, A. E. ..... 390 Main St., Op.
ESTABROOK, CHAS. T. ..... 10 Algonquin Road, OALR
GIBBY, HAROLD JAMES ..... 27 Elm St., ALR
GOODSPEED, HARRY B. ..... 36 Pleasant St., ALR
HOLZER, WM. F. ..... 36 Pleasant St., Op.
MESSIER, A. EUGENE ..... 390 Main St., OALR
MURPHY, WILLIAM E. ..... 390 Main St., ALR
RICE, JOHN E. ..... 390 Main St., OALR

MICHIGAN

ALMA

HOWELL, D. M. ..... Polasky Bldg., ALR

ALPENA

NEWTON, WM. B. ..... 201 N. Second Ave., OALR

ANN ARBOR

BARR, ALBERT S. ..... First Nat'l Bank Bldg., Op.
FRALICK, F. BRUCE ..... First Nat'l Bank Bldg., Op.
FURSTENBERG, A. C. ..... First Nat'l Bank Bldg., ALR
MAXWELL, JAMES H. ..... First Nat'l Bank Bldg., ALR

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SLEIGHT, R. D. ..... Security Nat'l Tower, Op.
WENCKE, CARL G. ..... Battle Creek Sanitarium, ALR

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SHERMAN, R. NEWTON ..... 400 Phoenix Bldg., OALR

BENTON HARBOR

WESTERVELT, H. O. ..... 239 Pipestone St., ALR

BIRMINGHAM

FERRIS, RALPH G. ..... 55 W. Maple Ave., ALR

DETROIT

AMBERG, EMIL ..... 622 Maccabee Bldg., ALR
BARNETT, SAUL E. ..... 25 Parsons St., ALR
BEATTIE, ROBERT ..... David Whitney Bldg., OALR
BEGLE, HOWELL L. ..... David Whitney Bldg., Op.
BENTLEY, NEIL I. ..... David Whitney Bldg., OALR
BERNSTEIN, E. J. ..... 10 Peterboro, OALR
BROWN, AUDREY O. ..... 5057 Woodward, OALR
CAMPBELL, DON M. ..... David Whitney Bldg., OALR
CAMPBELL, DUNCAN A. ..... David Whitney Bldg., OALR
CAMPBELL, MALCOLM D. ..... 269 Rowena St., OALR
CARTER, JOHN M. ..... David Whitney Bldg., ALR
CLIFFORD, T. P. ..... David Whitney Bldg., OALR
COHOE, D. A. ..... 13535 Woodward Ave., ALR
CROUSHORE, JAMES E. ..... 2501 W. Grand Blvd., ALR
DEFNET, WM. A. ..... 28 Adams Ave. West, ALR
DOWLING, HARVEY E. ..... Eaton Tower, Op.
FOWLER, WM. ..... Maccabee Bldg., OALR
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GLOWACKI, BEN FRANCIS ..... Maccabee Bldg., OALR
GOUX, RAYMOND SALOT ..... 545 David Whitney Bldg., OALR
GRANT, LEE E. ..... David Whitney Bldg., OALR
HALE, ARTHUR STANLEY ..... 1609 Eaton Tower, OALR
HAMMOND, ARTHUR ..... David Whitney Bldg., ALR
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HOWELL, DONALD MACLEAN .....David Whitney Bldg., ALR
HUGHES, RAY W. .....1501 David Whitney Bldg., OALR
KEATING, T. F. .....1042 Maccabee Bldg., OALR
KREUTZ, GEORGE C. .....Henry Ford Hospital, ALR
MCCLELLAND, CARL C. .....1857 David Whitney Bldg., OALR
MERCER, R. E. .....David Whitney Bldg., LR
MINER, S. G. .....2641 E. Jefferson Ave., ALR
MORTON, J. B. .....90 E. Warren Ave., OALR
MUNSON, F. T. .....2201 E. Jefferson Ave., ALR
O.HORA, B. A. .....3044 W. Grand Blvd., ALR
PARKER, WALTER R. .....David Whitney Bldg., Op.
PEIRCE, HOWARD W. .....David Whitney Bldg., ALR
PINO, RALPH H. .....David Whitney Bldg., Op.
POOS, E. E. .....Fisher Bldg., OALR
POTTER, WILLIS A. .....7450 Grand River Ave., ALR
RENAUD, G. L. .....510 Joseph Campan Ave., OALR
RYERSON, F. L. .....Fine Arts Bldg., OALR
SANDERSON, H. H. .....David Whitney Bldg., OALR
SCARNEY, HERMAN D. .....2501 W. Grand Blvd., Op.
SHURLY, BURT R. .....62 Adams Ave. W., ALR
SIMPSON, H. LEE .....David Whitney Bldg., ALR
SUMMERS, WM. S. .....1613 David Whitney Bldg., OALR
SUTHERLAND, JACOB MARION .....General Motors Bldg., ALR
WALKER, CHAS. C. .....Maccabee Bldg., Op.
WENDEL, J. S. .....744 David Whitney Bldg., ALR
WHITNEY, E. L. .....Henry Ford Hospital, ALR
WILLSON, W. W. .....2204 Eaton Tower, OALR
WOODWORTH, WILLIAM P. .....2994 E. Grand Blvd., OALR
WILKINSON, A. P. .....664 Fisher Bldg., ALR

FLINT

HUBBARD, WM. B. .....Dryden Bldg., ALR
STEVENSON, WM. W. .....Kresge Bldg., ALR

GRAND RAPIDS

DEAN, ALFRED .....308 Medical Arts Bldg., Op.
DEWAR, MURRAY M. .....Medical Arts Bldg., Op.
HEETDERKS, D. R. .....Medical Arts Bldg., ALR
LAIRD, ROBT. G. .....Metz Bldg., Op.
MCRAE, J. H. .....Medical Arts Bldg., Op.
MOLL, ARTHUR M. .....Powers Bldg., ALR
ROBERTSON, F. D. .....Ashton Bldg., OALR
ROLLER, LOUIS A. .....446 Paris Ave., S.E., Retired
SMITH, FERRIS .....Blodgett Medical Bldg., ALR
SNAPP, CARL F. .....Medical Arts Bldg., ALR

GROSSE POINTE VILLAGE

MERKEL, CHAS. C. .....279 Rivard Blvd., ALR

HOLLAND

HUIZINGA, J. G. .....Peoples State Bank Bldg., OALR

IONIA

SEDGWICK, O. W. ..... OALR

IRON MOUNTAIN

BOYCE, G. H. ..... Box 417, OALR

JACKSON

HARDIE, GEORGE C. ..... 290 W. Michigan Ave., ALR
MCGARVEY, WM. EDWARD ..... Jackson City Bank Bldg., ALR
WINTER, GEORGE E. ..... Rogers Bldg., OALR

KALAMAZOO

FULKERSON, C. B. ..... Room 403, 107 W. Michigan Ave., OALR
GRANT, FRED E. ..... Kalamazoo National Bank Bldg., OALR
WILBUR, EDWARD P. ..... American Bank Bldg., OALR

LANSING

BEHEN, WM. C. ..... 221 N. Capitol Ave., ALR
BURDICK, AUSTIN F. ..... 330 N. Capitol Ave., OALR
ELLIS, C. WARD ..... 215 N. Walnut St., ALR
MCGILLICUDDY, OLIVER B. ..... 124 W. Allegan St., ALR
WETZEL, JOHN O. ..... 124 W. Allegan St., Op.

MENOMINEE

JONES, WM. S. ..... 522 Sheridan Road, OALR

MT. CLEMONS

CROMAN, JOS. M., JR. ..... 115 Cass Ave., OALR

OWOSSO

POCHERT, ROLLAND C. ..... Matthews Bldg., ALR

PETOSKEY

CONWAY, WM. S. ..... 314½ Howard St., ALR

PONTIAC

RIKER, AARON D. ..... 1012 Riker Bldg., Op.

PORT HURON

FRASER, ROBERT C. ..... 940 Military St., ALR

ST. JOSEPH

MCDERMOTT, JOHN J. ..... 1821 Niles Ave., OALR

SAGINAW

CADY, F. J. ..... Peoples Bldg. and Loan Bldg., ALR
CORTOPASSI, ANDRE J. ..... Second Nat'l Bank Bldg., ALR
MCKINNEY, A. R. ..... 704 Second Nat'l Bank Bldg., Op.
SLACK, WALTER K. ..... 308 Eddy Bldg., ALR
WATSON, R. S. ..... 120 N. Michigan Ave., OALR

MINNESOTA

ALBERT LEA

FOLKEN, FRANK GARRETT ....Freeborn Co. Nat'l Bank Bldg., OALR

BRECKENRIDGE

RICE, CLARENCE PRENTICE .....OALR

CROOKSTON

MORLEY, GEORGE A. .....Crookston State Bank Bldg., Op.
OPPEGAARD, C. L. .....220 S. Broadway, ALR
PARSONS, J. G. .....Crookston Clinic, OALR

DULUTH

ARMSTRONG, E. LESLIE .....205 W. 2nd St., ALR
ATHENS, ALVIN G. .....Medical Arts Bldg., Op.
FELLOWS, M. FORDING .....Medical Arts Bldg., Op.
HILDING, ANDERSON C. .....Medical Arts Bldg., ALR
KNAPP, F. N. .....Medical Arts Bldg., OALR
SHASTID, T. H. .....202 W. Superior St., Op.
TILDERQUIST, D. L. .....Medical Arts Bldg., OALR
WINTER, JOHN A. .....205 W. Second St., OALR

FERGUS FALLS

COMBACHER, LEON C. .....101½ Lincoln Ave., W., Op.

HIBBING

MORSMAN, L. W. .....Morsman Clinic, Op.
SINAMARK, ANDREW .....205 Power Bldg., OALR

MINNEAPOLIS

BORGESON, E. J. .....Medical Arts Bldg., Op.
BROWN, EDWARD J. .....3027 Pleasant Ave., OALR
CAMP, WALTER E. .....Medical Arts Bldg., OALR
CAMPBELL, ROBERT A. .....520 La Salle Bldg., OALR
CLARK, H. S. .....Medical Arts Bldg., OALR
DAHL, ELMER O. .....Medical Arts Bldg., ALR
DORGE, RICHARD IRVING .....1111 Nicollet Ave., OALR
FINK, WALTER H. .....Medical Arts Bldg., OALR
HANSEN, ERLING W. .....Medical Arts Bldg., OALR
HOWARD, W. H. .....Physicians and Surgeons Bldg., OALR
KOLLER, LOUIS R. .....Medical Arts Bldg., ALR
LITCHFIELD, J. T. .....Medical Arts Bldg., OALR
LOOMIS, E. A. .....Medical Arts Bldg., OALR
MACNIE, J. S. .....Medical Arts Bldg., OALR
MATTHEWS, JUSTUS .....Medical Arts Bldg., LR
NEWHART, HORACE .....Medical Arts Bldg., ALR
OBERG, C. M. .....Syndicate Bldg., OALR
PATTERSON, W. E. .....Medical Arts Bldg., OALR
PHELPS, KENNETH A. .....Medical Arts Bldg., OALR

PRATT, FRED J. ..... Physicians and Surgeons Bldg., OALR
PRATT, JOHN A. ..... Physicians and Surgeons Bldg., OALR
REYNOLDS, J. S. ..... Medical Arts Bldg., OALR
RUCKER, CHAS. W. ..... Medical Arts Bldg., Op.
SCHWARTZ, VIRGIL JOSEPH ..... Medical Arts Bldg., ALR
SMITH, ARTHUR E. ..... Medical Arts Bldg., OALR
STROUT, E. S. ..... Medical Arts Bldg., OALR
STROUT, G. ELMER ..... 1009 Nicollet Ave., OALR
WATSON, JAMES A. ..... Medical Arts Bldg., OALR

NEW ULM

REINEKE, GEORGE F. ..... Box 137, OALR

RED WING

BRUSEGARD, JAS. F. ..... 414 Main St., Op.

ROCHESTER

ANDERSON, CARL M. ..... Mayo Clinic, ALR
BENEDICT, WILLIAM L. ..... Mayo Clinic, Op.
FIGI, F. A. ..... Mayo Clinic, LR
HEMPSTEAD, BERT E. ..... Mayo Clinic, ALR
LILLIE, HAROLD I. ..... Mayo Clinic, ALR
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PRANGEN, AVERY DE H. ..... Mayo Clinic, Op.
WAGENER, HENRY P. ..... Mayo Clinic, Op.

ST. CLOUD

WENNER, W. T. ..... St. Mary's Bldg., Op.

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BERRISFORD, PAUL D. ..... Lowry Medical Arts Bldg., OALR
BINGER, H. E. ..... Lowry Medical Bldg., OALR
BOECKMANN, EGIL ..... Lowry Medical Arts Bldg., OALR
BROWN, JOHN C. ..... Medical Arts Bldg., OALR
BURCH, FRANK E. ..... Hamm Bldg., Op.
CONNOR, CHAS. E. .... Lowry Medical Arts Bldg., 4th St. Sec., ALR
FOGARTY, CHAS. W. ..... 372 St. Peter St., OALR
GRANT, H. W. ..... Lowry Bldg., Op.
HOCHFILZER, JOHN J. ..... Lowry Medical Arts Bldg., ALR
LARSEN, CARL L. ..... 1027 Lowry Medical Bldg., OALR
LEAVENWORTH, R. O. ..... Lowry Medical Bldg., Op.
NELSON, ALLAN LOUIS ..... 830 Lowry Medical Arts Bldg., Op.
ROTHSCHILD, HAROLD J. ..... Lowry Bldg., OALR
SHELLMAN, J. L. ..... Lowry Medical Arts Bldg., OALR
WHEELER, MERRITT W. ..... 1027 Lowry Bldg., OALR
WOLD, K. C. ..... Lowry Medical Arts Bldg., OALR

WINONA

RISSER, EDWARD D. ..... 310 Choate Bldg., OALR
WALKER, GEORGE H. ..... 172 Main St., OALR

WORTHINGTON

STANLEY, C. R. ..... 508 Tenth St., ALR

MISSISSIPPI

GREENVILLE

MONTGOMERY, D. CAMERON ..... Leyser Bldg., ALR

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HAGEMAN, VAN DYKE ..... Lamar Life Bldg., ALR
HARRIS, ROBIN ..... Lamar Life Bldg., ALR

NATCHEZ

GAUDET, LUCIAN S. ..... 405 Franklin St., OALR

OXFORD

GUYTON, B. S. ..... 512 Van Buren Ave., OALR

VICKSBURG

EDWARDS, C. J. ..... First National Bank Bldg., OALR
JONES, EDLEY H. ..... 1301 Washington St., OALR

MISSOURI

CARTHAGE

POST, WINFRED B. ..... 304 Grant St., OALR
POWERS, EVERETT ..... 305 S. Main St., OALR

COLUMBIA

SNEED, CARL M. ..... Guitar Bldg., OALR

EXCELSIOR SPRINGS

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HANNIBAL

HORNBACK, GEORGE A. ..... 500 Broadway, ALR

KANSAS CITY

ALLEN, CHAS. E. ..... 712 Waldheim Bldg., OALR
ALTRINGER, A. N. ..... Professional Bldg., ALR
BEAL, HOMER A. ..... 1002 Argyle Bldg., ALR
BEIL, J. W. ..... Bryant Bldg., OALR
BELLOWS, GEORGE E. ..... 1500 Professional Bldg., Op.
BLACK, W. BYRON ..... Professional Bldg., ALR
BLAKESLEY, T. S. ..... Professional Bldg., OALR
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CURRAN, E. J. ..... 1805 Fed. Res. Bank Bldg., Op.
DAVIS, HARRY B. ..... Bryant Bldg., OALR
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FOSTER, HAL ..... 1426 Professional Bldg., ALR
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HOWARD, J. W. ..... Professional Bldg., OALR
KEITH, W. E. ..... Professional Bldg., OALR
KNIGHT, JOHN S. ..... Professional Bldg., ALR
LANING, J. HALCOMBE ..... 907 Rialto Bldg., ALR
LEMOINE, A. N. ..... Rialto Bldg., Op.
LORIE, ALVIN J. ..... Commerce Bldg., ALR
LUX, PAUL ..... 2006 Bryant Bldg., ALR
MCALESTER, ANDREW W., JR. ..... Bryant Bldg., Op.
MCALESTER, ANDREW W., 3RD ..... Bryant Bldg., Op.
MCCARTY, VIRGIL W. ..... Professional Bldg., OALR
MCKEE, JOSEPH W. ..... Professional Bldg., Op.
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ROBERTS, S. E. ..... Professional Bldg., ALR
SHUMATE, D. L. ..... 500 Argyle Bldg., OALR
SIMPSON, M. B. ..... Professional Bldg., ALR
THOMASON, H. E. ..... Professional Bldg., ALR
TEALL, RAYMOND E. ..... Professional Bldg., ALR
YAZEL, HERMAN E. ..... 733 Rialto Bldg., OALR

POWERSITE

COFFELT, THEODORE A. ..... OALR

ST. JOSEPH

FORGRAVE, L. ROBERT ..... 204 Logan Bldg., OALR
MINTON, WM. H. ..... Carby Bldg., OALR
PROUD, WILLARD C. ..... Phys. and Surg. Bldg., OALR

ST. LOUIS

ALDEN, ARTHUR M. ..... Frisco Bldg., ALR
ALVIS, BENNETT Y. ..... 308 N. 6th St., Op.
ARBUCKLE, MILLARD F. ..... 539 N. Grand Ave., ALR
BARDENHEIER, F. G. A. ..... Frisco Bldg., ALR
BEISBARTH, CARL C. ..... 3720 Washington Ave., Op.
BIRSNER, LOUIS J. ..... Missouri Bldg., ALR
BLACK, W. D. ..... Metropolitan Bldg., ALR
BOEMER, L. C. ..... 3720 Washington Blvd., ALR
BROWN, CLYDE O. ..... 3720 Washington Blvd., ALR
BURNS, S. S. ..... Missouri Theater Bldg., ALR
CALHOUN, JAMES GRAHAM ..... Metropolitan Bldg., Op.
CHARLES, JOS. W. ..... 3720 Washington Ave., Op.
CONE, ALFRED J. ..... 4932 Maryland St., ALR
COSTEN, J. B. ..... Beaumont Medical Bldg., ALR

DEAN, L. W. ..... Medical School, Washington Univ., ALR
DOBBS, OTTO ROSS ..... 2743 N. Grand Ave., ALR
DYER, C. P. ..... 408 Humboldt Bldg., Op.
EBER, CARL THEODORE ..... 1006 Carlton Bldg., Op.
EIMER, CHARLES EUGENE ..... 316 Lister Bldg., ALR
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FINEBERG, MAXWELL ..... Missouri Theatre Bldg., ALR
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GOLDSTEIN, M. A. ..... 4574 W. Papin St., ALR
GREEN, JOHN ..... 3720 Washington Blvd., Op.
GREENE, M. L. ..... Missouri Theater Bldg., Op.
GUGGENHEIM, L. K. ..... 6227 Rosebury, ALR
GUNDELACH, C. A. ..... 825 University Club Bldg., ALR
HANSEL, FRENCH KELLER ..... 634 No. Grand Ave., ALR
HARDESTY, J. F. ..... 706 Missouri Theater Bldg., Op.
HARDY, W. F. ..... Humboldt Bldg., Op.
HILDRETH, H. ROMMEL ..... Metropolitan Bldg., Op.
HOBART, CARL ..... Metropolitan Bldg., Op
HOOSS, C. H. A. ..... 3115 S. Grand Blvd., Op.
HOURN, GEORGE E. ..... 513 University Club Bldg., ALR
HOWARD, HARVEY J. ..... Park Plaza Hotel, Op.
JACOBS, MAX W. ..... Missouri Theater Bldg., Op.
JAMES, WM. M. ..... 508 N. Grand Ave., Op.
JONES, VINCENT L. ..... 1242 Missouri Theater Bldg., OALR
KELLEY, ISAAC D., JR. ..... 3720 Washington Blvd., ALR
LAMB, HARVEY D. ..... Metropolitan Bldg., ALR
LEAVY, C. A. ..... Metropolitan Bldg., ALR
LUEDDE, W. H. ..... Metropolitan Bldg., Op.
LYMAN, HARRY W. ..... 308 N. 6th St., ALR
MCMAHON, BERNARD JOHN ..... Missouri Theater Bldg., ALR
MASON, ROY EARL ..... Frisco Bldg., Op.
MILLER, H. ED. ..... 728 Missouri Bldg., ALR
MYERS, EUGENE LEE ..... Wall Bldg., ALR
NEMOURS, PAUL ROLAND ..... 3142 Franklin Ave., ALR
PAYNE, R. J. ..... University Club Bldg., ALR
PELZ, MORT D. ..... Missouri Bldg., ALR
POOS, GROVER H. ..... 607 N. Grand Ave., OALR
POST, LAWRENCE T. ..... Metropolitan Bldg., Op.
POST, M. HAYWARD ..... Metropolitan Bldg., Op.
POTTER, ALLEN B. ..... 5068 Thologan, ALR
PROETZ, A. W. ..... Beaumont Bldg., ALR
REIM, HUGO ..... 417 Metropolitan Bldg., Op.
RYAN, L. M. ..... Wall Bldg., ALR
SAUER, W. E. ..... Beaumont Medical Bldg., ALR
SCHLUER, E. P. ..... 508 N. Grand Ave., ALR
SCHOLZ, R. P. ..... Metropolitan Bldg., ALR
SCHWARTZ, FREDERICK O. ..... 508 N. Grand Blvd., Op.
SENSENEY, EUGENE T. ..... 316 Lister Bldg., ALR
SHAHAN, W. E. ..... Metropolitan Bldg., Op.

SHOEMAKER, W. A. .....1006 Carleton Bldg., Op.
SIMON, FREDERICK C. .....914 Arcade Bldg., ALR
SMIT, WM. M. .....5285 Washington Bvld., ALR
TOOKER, C. W. .....Carleton Bldg., Op.
WESTLAKE, SAMUEL B. .....Beaumont Med. Bldg., ALR
WIENER, MEYER .....900 Carleton Bldg., Op.
WOOD, V. VISSCHER .....Beaumont Medical Bldg., ALR
WOODRUFF, FREDERICK E. .....Metropolitan Bldg., Op.

SEDALIA

LOVE, JOHN G. .....Ilgenfritz Bldg., OALR
TITSWORTH, GUY .....111 W. 4th St., OALR

SPRINGFIELD

BAILEY, HAROLD .....Landers Bldg., OALR
KLINGNER, THOS. O. .....Medical Arts Bldg., OALR
SMITH, CARLIE SOUTER .....214 N. Jefferson Ave., OALR

TRENTON

AMBROSE, ELMER C. .....910 Main St., OALR

MONTANA

ANACONDA

DUNLAP, LAWRENCE G. .....101 Main St., OALR

BILLINGS

MORLEDGE, ROY V. .....Hart Albin Bldg., ALR

BUTTE

DONOVAN, JOHN A. .....507 Phoenix Bldg., OALR
MORSE, A. W. .....507 Phoenix Bldg., OALR
POTTER, PETER .....Motor Clinic Bldg., OALR

GREAT FALLS

COULTER, CHAS. F. .....First Nat'l Bank Bldg., OALR

HAVRE

BRIGGS, F. W. .....Masonic Temple, OALR

ROUNDUP

LEWIS, GEORGE A. .....213 Main St., OALR

NEBRASKA

COLUMBUS

CAMPBELL, C. H. .....Maxwell and Bratt Bldg., OALR
KOEBBE, EZRA EDWIN .....Columbus Bank Bldg., ALR

GRAND ISLAND

RIGG, JAMES P. .....217½ N. Pine St., ALR

HASTINGS

MARTIN, ROY DAVID ..... Foote Bldg., ALR

KEARNEY

FOX, CHAS. H. ..... 5 West 21st St., OALR

LINCOLN

BLACK, PAUL ..... Stuart Bldg., Op.
BROOKS, EARL B. ..... 939 Stuart Bldg., OALR
FURGASON, A. P. ..... Richards Block, OALR
HOMPES, J. J. ..... Sharp Bldg., OALR
SANDERSON, DAVID D. ..... Stuart Bldg., OALR
THOMAS, JAMES W. ..... Sharp Bldg., OALR
ZEMER, S. G. ..... Stuart Bldg., OALR

NEBRASKA CITY

STONECYPHER, D. D. ..... 107 S. 9th St., ALR

NORFOLK

GADBOIS, ARTHUR E. ..... DeLay Nat'l Bank Bldg., OALR

OMAHA

BUSHMAN, L. B. ..... 631 City Nat'l Bank Bldg., OALR
CALLFAS, W. F. ..... 1620 Medical Arts Bldg., ALR
CASSIDY, W. A. ..... Medical Arts Bldg., ALR
DAVIS, J. CALVIN ..... 425 Aquila Court, OALR
FAIER, SAMUEL Z. ..... Medical Arts Bldg., ALR
HANEY, WM. P. ..... 1500 Medical Arts Bldg., OALR
JUDD, DELBERT K. ..... Medical Arts Bldg., ALR
JUDD, J. H. ..... Medical Arts Bldg., Op.
KNODE, A. R. ..... 708 Medical Arts Bldg., ALR
KULLY, HERMAN E. ..... Medical Arts Bldg., ALR
POTTER, GEORGE B. ..... Medical Arts Bldg., OALR
POTTS, J. B. ..... Medical Arts Bldg., ALR
ROMONEK, PHILIP L. ..... Medical Arts Bldg., OALR
RUBENDALL, C. ..... Medical Arts Bldg., OALR
STOKES, WM. H. ..... Medical Arts Bldg., Op.
SWAB, CHARLES M. ..... Medical Arts Bldg., Op.
UREN, C. T. ..... 537 City National Bank Bldg., ALR
WHERRY, W. P. ..... 1500 Medical Arts Bldg., OALR

NEVADA

RENO

FULLER, JOHN ANDRE ..... First Nat'l Bank Bldg., OALR

NEW HAMPSHIRE

NASHUA

KITTREDGE, FRANK E. ..... Masonic Temple, ALR
MARCOTTE, RAYMOND H. ..... 215A Main St., ALR
NUTTER, C. F. ..... 16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

BAESEMAN, R. WINFIELD.....501 Grand Ave., ALR
FISHER, JAMES A. .....Jersey Central Bldg., OALR
UPHAM, HELEN F. .....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. COULTER .....124 S. Illinois Ave., ALR
MCGIVERN, C. S. .....805 Pacific Ave., ALR
OLMSTEAD, WILLIAM DEF. .....515 Grammercy Place, Op.

BLOOMFIELD

MOORE, JOHN D. .....6 Washington St., OALR

BOUND BROOK

LEVY, ABRAM .....507 Church St., Op.

CAMDEN

KLINE, ORAM R. .....414 Cooper St., ALR
SHEMELEY, WILLIAM G. .....7 Haddon Ave., OALR
SHIPMAN, JAMES S. .....542 Cooper St., Op.
SHOPE, PIERCE .....511 Cooper St., Op.

EAST ORANGE

BUVINGER, CHARLES W. .....50 Washington St., OALR

ELIZABETH

KAPP, CARL G. .....440 Westminster Ave., ALR

ENGLEWOOD

LITTWIN, CHARLES .....33 E. Palisade Ave., Op.

HACKENSACK

BERKE, RAYNOLD N. .....430 Union St., Op.
GREENFIELD, WILLIAM JOHN .....50 Anderson St., ALR

JERSEY CITY

PYLE, WALLACE .....15 Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K. .....96 Third Ave., OALR

NEWARK

BARKHORN, CHAS. W. .....223 Roseville Ave., ALR
CARDWELL, EDGAR PARMELE .....47 Central Ave., ALR
CHATTIN, J. FRANKLIN .....671 Broad St., Op.
EAGLETON, WELLS P. .....15 Lombardy St., ALR

HAHN, WM. H. .....15 Lombardy St., Op.
HUGHES, L. W. .....965 Broad St., OALR
HURFF, J. WALLACE .....86 Washington St., OALR
KEIM, WILLIAM F. .....25 Roseville Ave., ALR
O'CONNOR, D. F. .....671 Broad St., OALR
ORTON, HENRY B. .....24 Commerce St., ALR
PEER, L. A. .....965 Broad St., ALR
QUINBY, W. O'G. .....14 James St., OALR
SHERMAN, A. RUSSELL .....671 Broad St., Op.
SHERMAN, ELBERT S. .....671 Broad St., Op.
WOOD, EARL LEROY .....172 Roseville Ave., ALR
ZEHNDER, A. CHARLES .....188 Roseville Ave., Op.

ORANGE

EMERSON, LINN .....Metropolitan Bldg., Op.

PATERSON

ATWOOD, ED. A. .....360 Park Ave., ALR

PERTH AMBOY

COHEN, NATHEN B. .....232 State St., ALR
MANN, J. J. .....255 State St., ALR

PLAINFIELD

CHILDERS, ROBERT JEFFERSON .....604 Park Ave., ALR
VAN HORN, A. F. .....514 Central Ave., ALR

RIDGEWOOD

DENISON, WARD C. .....123 Prospect St., ALR

SOUTH ORANGE

MILLER, JOSEPH A. .....364 Prospect St., ALR

SUMMIT

BURRITT, NORMAN W. .....30 Beechwood Road, ALR
SLY, JOHN L. .....382 Springfield Ave., ALR

TRENTON

ADAMS, C. F. .....34 W. State St., OALR

UNION CITY

SACCO, ANTHONY G. .....440 New York Ave., OALR

WEST NEW YORK

SELINGER, SAMUEL .....413 Sixteenth St., OALR

NEW MEXICO

ALBUQUERQUE

BREHMER, HARRISON L. .....First Nat'l Bank Bldg., OALR

FORT BAYARD

GILL, ARTHUR E. .....U. S. Veterans' Hospital, OALR

ROSWELL

GRISWOLD, GEORGE WAYNE .....211 W. 3rd St., ALR

SANTA FE

BARTON, WM. C. .....104½ W. San Francisco St., Op.

NEW YORK

ALBANY

BEDELL, ARTHUR J. .....344 State St., Op.
DOWLING, J. IVIMEY .....116 Washington Ave., OALR
HINMAN, E. E. .....146 Washington Ave., ALR
JUDGE, HARRY V. .....46 Willett St., Op.
SIMON, EMANUEL .....238 Lark St., ALR
TEBBUTT, H. K. .....240 State St., ALR

AUBURN

BULLARD, C. W. .....120 Genesee St., ALR

BATAVIA

WILSON, ROBERT G. .....433 E. Main St., ALR

BINGHAMTON

KINNE, SANFORD HALSEY .....73 Main St., ALR
RANDALL, HIRAM .....81 Main St., ALR
SMITH, WINDSOR R. .....67 Front St., ALR
WATSON, H. DE WITT .....151 Front St., OALR

BRONXVILLE

MCKEOWN, HUGH S. ..... Op.

BROOKLYN

AGAN, WM. B. .....510 Third St., Op.
ALBRIGHT, P. M., LT. COMDR. (M.C.)U.S.N..U. S. Naval Hosp., OALR
ANDREW, JAS. H. .....163 Hancock St., Op.
BEHRENS, ALVIN .....9 Prospect Park West, Op.
BERGMANN, MILTON B. .....959 Bushwick Ave., Op.
BERNSTEIN, P. F. .....1265 President St., ALR
BROOKS, MORRIS J. .....455 Ocean Ave., ALR
BRUNO, JOSEPH M. L. .....31 Jefferson St., Op.
BUONAGURO, MICHAEL J. .....589 Lorimer St., Op.

COHEN, WILLIAM .....730 Eastern Parkway, Op.
CUTINO, R. M. .....165 Clinton St., Op.
DECOSTE, S. H. .....1179 Dean St., ALR
EBELING, WM. B. .....54 Greene Ave., Op.
EVANS, J. N. .....23 Schermerhorn St., Op.
FEASTER, HENRY J. .....461 77th St., ALR
FELDMAN, LOUIS A. .....259 New York Ave., Op.
FINE, ABRAHAM .....1505 Ocean Ave., Op.
GOLDEN, MATTHEW G. .....1 Nevins St., ALR
GOLDING, JOSEPH E. .....170 Hancock St., Op.
GREENFIELD, SAMUEL D. .....169 New York Ave., ALR
GRIFFIN, EDWIN A. .....165 Hancock St., ALR
HARGITT, CHARLES A. .....17 Schermerhorn St., Op.
HOLZBERGER, JOHN C. .....7005 Freshpond St., ALR
HULL, ALLEN .....1847 Benson Ave., Op.
JAMESON, P. CHALMERS .....139 Montague St., Op.
KAPLAN, SAMUEL .....1 Nevins St., ALR
KRIMSKY, EMANUEL .....754 Eastern Parkway, Op.
KRIMSKY, JOSEPH .....302 Empire Blvd., OALR
KRUSKAL, I. D. .....101 Lafayette Ave., Op.
LEIBOWITZ, PHILIP .....135 E. Parkway, ALR
LEVITT, JESSE M. .....734 Ocean Ave., Op.
LLOYD, RALPH I. .....14 Eighth Ave., Op.
LOMBARDO, MELCHIORE .....142 Joralemon St., Op.
LUPO, CARL W. .....7425 Ridge Blvd., ALR
MENGER, LOUIS CHARLES .....202 Weirfield St., ALR
MOEHLE, WALTER .....15 Schermerhorn St., Op.
MOORHEAD, ROBERT L. .....125 Remsen St., ALR
NOVA, JULES M. .....349 Adelphi St., Op.
O'BRIEN, G. R. .....307 Sterling Place, ALR
OHLY, JOHN H. .....22 Schermerhorn St., Op.
RICHMAN, FRANCES .....522 Ocean Ave., Op.
ROGERS, ROBERT M. .....680 St. Johns Pl., Op.
SCHILLINGER, RAPHAEL .....50 Plaza St., ALR
SHERMAN, MAJ. A. EDWARD, N. Y. Port of Embarkation, Office of Port Surgeon, 58 St. and 1st Ave., OALR
SPARBER, HERMAN .....4701 Fifteenth Ave., Op.
STEINBUGLER, WM. F. C. .....515 Ocean Ave., Op.
THOMASHEFSKY, M. .....135 Eastern Parkway, ALR
WECHSLER, A. H. .....340 New York Ave., ALR
WEETH, CHAS. R. .....37 Remsen St., ALR
DE YOANNA, ALFRED A. .....111 Pierrepont St., ALR

BUFFALO

ANDREWS, H. D. .....33 Allen St., Op.
BENNETT, ARTHUR G. .....150 N. Pearl St., Op.
BEYER, LOUIS J. .....449 Delaware Ave., ALR
BLAAUW, E. E. .....190 Ashland Ave., Op.
BOZER, HERRMANN EUGENE .....468 Delaware Ave., ALR

BROWN, C. M. .....510 Delaware Ave., ALR
COTT, C. C. .....333 Linwood Ave., ALR
COWPER, H. W. .....543 Franklin St., Op.
EDSON, R. A. .....957 Delaware Ave., Op.
FAIRBAIRN, JOHN F. .....925 Delaware Ave., ALR
FLAGG, J. D. .....158 Anderson Place, Op. A
GLOSSER, HERBERT H. .....448 Franklin St., Op.
KOENIG, IVAN J. .....40 North St., Op.
LE WIN, THURBER .....112 Linwood Ave., Op.
LEWIS, F. PARK .....454 Franklin St., Op.
LUHR, A. F. .....40 W. North St., Op.
MCKEE, OTTO S. .....83 Bryant St., ALR
MARCH, CLARA A. .....465 Ashland Ave., Op.
PHILLIPS, WM. LINTON .....131 Linwood Ave., Op.
RIWCHUN, MEYER H. .....468 Delaware Ave., Op.
TROTTER, HOMER A. .....40 North St., ALR
WEED, HARRY M. .....196 Linwood Ave., Op.
WURTZ, W. J. M. .....333 Linwood Ave., ALR

CORTLAND

HIGGINS, R. P. .....20 Court St., OALR

DUNKIRK

JACKLE, ARTHUR F. .....63 E. 4th St., Op.

ELMIRA

GLOVER, ARTHUR C. .....311 W. Church St., OALR

FLUSHING

GOODE, EDWARD ALLEN .....143-28 Sanford Ave., ALR

GENEVA

SPENGLER, JOHN ARTHUR .....423 S. Main St., Op.

GLENS FALLS

BUCK, CLAIR A. .....191 Glen St., ALR
FIELDING, F. G. .....70 Ridge St., ALR

JAMAICA

BENDER, M. S. .....88-05 150th St., ALR
KEELER, LANSING H. .....89-04 148th St., ALR
MILLER, SAUL .....8805 150th St., Op.

JAMESTOWN

REGER, H. S. .....Physicians Bldg., OALR

LITTLE FALLS

NEWTON, MORRIS H. .....Burrell Bldg., Op.

LOCKPORT

PARKER, HARRY L. .....88 East Ave., ALR
RINGUEBERG, EUGENE N. S. .....13 Main St., Op.

LONG ISLAND — HEMPSTEAD

COON, EUGENE H. .....131 Fulton Ave., ALR
HUGHES, WENDELL L. .....Professional Bldg., Op.
HULKA, J. H. .....30-28 36th St., OALR

MIDDLETOWN

HICKS, WM. J. .....15 King St., ALR
NELLER, WALTER I. .....121 Wickham Ave., Op.

MT. VERNON

KEEFE, WM. JOHN .....110 E. Sidney Ave., ALR
THOMSON, J. J. .....531 E. Lincoln Ave., ALR

NEWBURGH

BROWNING, BIRT LEE .....208 Grand St., ALR

NEW ROCHELLE

BECK, A. L. .....421 Huguenot St., OALR
HETRICK, L. E. .....260 North Ave., ALR
WOODRUFF, WALTER STUART .....421 Huguenot St., ALR

NEW YORK CITY

AEBLI, RUDOLF .....30 E. 40th St., Op.
ALLAN, WM. B. .....410 Park Ave., Op.
ALMOUR, RALPH .....51 W. 73rd St., ALR
AUERBACH, JULIUS .....1016 Fifth Ave., ALR
BALLIN, M. J. .....20 E. 76th St., ALR
BASSEN, ED. J. .....654 Madison Ave., Op.
BEACH, B. S. .....140 W. 86th St., Op.
BENDER, M. S. .....30 E. 40th St., ALR
BERENS, CONRAD .....35 E. 70th St., Op.
BERKE, RAYNOLD N. .....30 W. 59th St., Op.
BERLINER, M. L. .....57 W. 57th St., Op.
BILCHICK, EDWIN B. .....470 Park Ave., ALR
BOYES, T. L. .....654 Madison Ave., Op.
BRANIGAN, C. HUGH .....399 Park Ave., Op.
BRUENING, PAUL H. .....133 E. 58th St., ALR
BRUCE, G. M. .....635 W. 165th St., Op.
BRUDER, JOSEPH .....57 W. 57th St., OALR
BULLWINKEL, H. GRIFFIN .....30 E. 76th St., ALR
BUNNELL, CHESTER WM. .....121 E. 60 th St., ALR
BURMAN, HERMAN J. .....1235 Grand Concourse, ALR
CARTER, W. W. .....140 E. 54th St., ALR
CASTROVIEJO, RAMON .....635 W. 165th St., Op.

COHEN, MARTIN .....29 E. 64th St., Op.
COLBERT, ROBERT MARTIN .....110 W. 55th St., ALR
CONBOY, JOHN ED. .....2257 University Ave., Op.
CORYA, HAROLD W. .....30 E. 40th St., ALR
COTT, D. N. .....25 Central Ave., St. George, Staten Isl., ALR
CRACOVANER, ARTHUR J. .....103 E. 78th St., ALR
CUNNING, DANIEL S. .....145 E. 54th St., OALR
DAVIDSON, MORRIS .....80 Centre St., Dept. of Labor, Op.
DAVIS, A. EDWARD .....40 E. 61st St., Op.
DAVIS, G. E. .....1060 Fifth Ave., OALR
DEPPING, CHAS. WM. .....19 W. 54th St., ALR
DIXON, G. S. .....1150 Fifth Ave., R
DLUGASCH, LOUIS .....110 E. 36th St., ALR
DOHERTY, W. B. .....150 W. 55th St., Op.
DUNNINGTON, J. H. .....30 W. 59th St., Op.
ELLIS, Z. H. .....58 E. 65th St., Op.
FERNLUND, GEORGE BYRON .....8577 112th St., Richmond Hill, OALR
FITZGERALD, FRED J. C. .....424 Madison Ave., Op.
FOSTER, HAROLD .....9205 — 35th Ave., Jackson Heights, ALR
FOWLER, ROBERT HENRY .....149 E. 62nd St., ALR
FOWLKES, JOHN WINSTON .....30 W. 59th St., ALR
FRANKEL, EDWARD, JR. .....217 E. 17th St., ALR
FREY, W. G., JR. .....121 E. 60th St., Op.
FRIEDMAN, BENJAMIN .....6 W. 77th St., Op.
GATEWOOD, W. L. .....330 Park Ave., ALR
GERSTLEY, MANFRED JOS. .....30 E. 40th St., ALR
GOLDMAN, A. MILTON .....121 E. 60th St., Op.
GOLDMAN, H. G. .....57 W. 58th St., ALR
GOLDSTEIN, ISADORE .....1095 Park Ave., Op.
GOTTLIEB, MARK J. .....2 W. 87th St., ALR
GRAEBNER, HERBERT .....45 Park Ave., ALR
GRESSER, EDWARD BELLAMY .....816 Fifth Ave., Op.
GRUSHLOW, ISRAEL .....271 Central Park, W., ALR
GUY, LOREN P. .....116 E. 62nd St., Op.
HALLETT, G. DEWAYNE .....274 W. 86th St., Op.
HARDY, LEGRAND H. .....30 E. 40th St., Op.
HARRIS, THOMAS JEFFERSON .....104 E. 40th St., ALR
HARTSHORNE, ISAAC .....30 W. 59th St., Op.
HASKIN, W. H. .....300 Park Ave., ALR
HAYS, HAROLD M. .....133 E. 58th St., ALR
HETRICK, L. E. .....40 E. 61st St., ALR
HIRSCH, CAESAR .....667 Madison Ave., ALR
HORN, WALTER L. .....1040 Park Ave., ALR
HURD, L. M. .....39 E. 50th St., ALR
HUNT, WESTLEY MARSHALL .....33 E. 68th St., ALR
HUTCHINSON, A. T. .....30 W. 59th St., ALR
IMPERATORI, C. J. .....108 E. 38th St., ALR
INGERMAN, S. M. .....418 Central Park, W., OALR
JACOBS, S. M. .....1749 Grand Concourse, OALR
JARECKY, HERMAN .....120 W. 86th St., OALR

JEROME, WM. Z. ..... 436 Fort Washington Ave., ALR
JOHNSON, THOMAS HOLLAND ..... 30 W. 59th St., Op.
JONES, DAVID H. ..... 140 E. 54th St., LR
JONES, MARVIN F. ..... 121 E. 60th St., ALR
JOSEPHSON, EMANUEL M. ..... 108 E. 31st St., ALR
JUDD, H. B. ..... 40 E. 61st St., ALR
KELLY, JOSEPH DOMINIC ..... 140 E. 54th St., OALR
KEND, LEE ..... 405 Park Ave., ALR
KEY, BEN WITT ..... 100 W. 59th St., Op.
KING, JAMES J. ..... 133 E. 58th St., ALR
KIRBY, D. B. ..... 780 Park Ave., Op.
KIRKBRIGHT, JAMES C. ..... 40 E. 61st St., ALR
KNAPP, ARNOLD ..... 10 E. 54th St., Op.
KNAPP, ARTHUR A. ..... 133 E. 58th St., Op.
KNIGHTON, W. S. ..... 40 E. 61st St., Op.
KOLLER, CARL ..... 30 E. 58th St., Op.
KOPETZKY, S. J. ..... 51 W. 73rd St., ALR
KRUG, ERNEST F. ..... 988 Fifth Ave., Op.
LAMBERT, ROBERT K. ..... 1010 Fifth Ave., Op.
LA VIGNE, A. A. ..... 471 Park Ave., OALR
LEVINE, JOSEPH ..... 105 E. 63rd St., Op.
LITTWIN, CHAS. ..... 550 Park Ave., Op.
LOEWE, WALTER R. ..... 1114 Madison Ave., Op.
LUBMAN, MAX ..... 118 E. 93rd St., ALR
MACDONALD, PHILIP G. ..... 115 E. 61st St., ALR
MACNIE, JOHN P. ..... 635 W. 165th St., Op.
MCAULIFFE, GERVAIS W. ..... 110 W. 55th St., ALR
MCDANNALD, C. E. ..... 100 W. 59th St., Op.
MCDONALD, DENNIS J. ..... 17 E. 38th St., ALR
MCKEOWN, HUGH S. ..... 30 W. 59th St., Op.
MATUSOW, ABRAHAM P. ..... 1100 Grand Concourse, ALR
MAY, CHAS. H. ..... 698 Madison Ave., Op.
MAYBAUM, J. L. ..... 1070 Park Ave., ALR
MEEK, R. E. ..... 729 Park Ave., Op.
MEIERHOF, E. L. (Retired) ..... care Guaranty Trust Co., OALR
MILLER, JOSEPH WILLIAM ..... 305 E. 17th St., ALR
MINSKY, HENRY ..... 1 W. 86th St., Op.
MITTENDORF, A. D. ..... 399 Park Ave., Op.
MITTENDORF, W. KEITH ..... 115 E. 53rd St., Op.
MORRISON, WALLACE ..... 39 E. 50th St., ALR
MOYLE, EUGENE H. ..... 1088 Park Ave., ALR
MYERSON, M. C. ..... 136 E. 64th St., ALR
NEAL, PHIL HAWKINS ..... 111 E. 75th St., ALR
NISSELSON, MAX ..... 754 Beck St., ALR
NORTHINGTON, PAGE ..... 20 E. 53rd St., ALR
OAST, SAMUEL P., JR. ..... 30 E. 40th St., Op.
PALMER, ARTHUR ..... 667 Madison Ave., ALR
PAYNE, B. F. ..... 35 E. 70th St., Op.
PETERSON, R. A. ..... 150 Fifth Ave., OALR
PFEIFFER, RAYMOND L. ..... 635 W. 165th St., Op.

REESE, ALGERNON B. ..... 73 E. 71st St., Op.
RISCH, OTTO C. ..... 104 E. 40th St., ALR
ROSENBAUM, MORRIS ..... 61 W. 88th St., Op.
ROSENBLUTH, MICHAEL ..... 1018 E. 163rd St., ALR
SACKIN, DAVID ..... "St. Moritz," 50 Central Park South, ALR
SAMUELS, BERNARD ..... 57 W. 57th St., Op.
SCAL, JOSEPH COLEMAN ..... 27 W. 96th St., ALR
SCHECHTER, SOLOMON ..... 1749 Grand Concourse, ALR
SCHEER, HENRY M. ..... 522 West End Ave., ALR
SCHILLER, A. NOAH ..... 225 Central Park West, ALR
SCHLIVEK, KAUFMAN ..... 1016 Fifth Ave., Op.
SCHMIDT, OTTO V. M. ..... 111 E. 61st St., ALR
SCHOENBERG, MARK J. ..... 667 Madison Ave., Op.
SCHUGT, HENRY P. ..... 30 E. 40th St., ALR
SCHWARTZ, A. A. ..... 7 W. 71st St., ALR
SCHWARTZ, LEO, JR. ..... 1 W. 73rd St., ALR
SETTEL, NATHAN ..... 10 W. 82nd St., ALR
SEWARD, J. A. ..... 115 E. 61st St., ALR
SITCHEVSKA, OLGA L. ..... 160 W. 87th St., Op.
SMITH, JAS. M. ..... 123 E. 53rd St., ALR
SOMBERG, J. S. ..... 470 Park Ave., Op.
STARK, JESSE B. ..... 45 Park Ave., Op.
STRAATSMA, CLARENCE R. ..... 899 Park Ave., ALR
STURGES, LEIGH F. ..... 121 E. 60th St., ALR
TUSAK, E. A. ..... 115 E. 90th St., Op.
WACHSBERGER, ALFRED ..... 51 W. 73rd St., ALR
WEEKS, WEBB WILLIAM ..... 20 E. 53rd St., Op.
WEINSTEIN, A. J. ..... 244 W. 74th St., ALR
WEXLER, DAVID ..... 179 E. 79th St., Op.
WHEELER, J. M. ..... 635 W. 165th St., Op.
WHEELER, MAYNARD C. ..... 30 W. 59th St., Op.
WHELAN, WILLIAM F. ..... 2015 Walnut St., ALR
WHITE, JAMES W. ..... 15 Park Ave., Op.
WIENER, ALFRED ..... 550 Park Ave., OALR
WINCOR, HENRY G. ..... 1882 Grand Concourse, Op.
WOOD, ED. C. ..... 405 Park Ave., Op.
WOODMAN, DEGRAAF ..... 156 E. 37th St., ALR

NIAGARA FALLS

PRICE, NORMAN W. ..... 445 Third St., OALR
ROOKER, A. M. ..... 203 Gluck Bldg., ALR

NYACK

TOMS, S. W. S. ..... 120 S. Broadway, OALR

ONEIDA

CROCKETT, R. L. ..... 302 Main St., Op.

ONEONTA

HALL, EDWIN P. .....76 Chestnut St., ALR

OSSINING

NEWLANDS, WM. A. .....16 Maple Place, ALR

PLATTSBURG

SCHNEIDER, ANTON S. .....56 Court St., Op.A

POUGHKEEPSIE

KRIEGER, WM. A. .....103 Hooker Ave., OALR
VOORHEES, EARLE W. .....25 Market St., ALR

ROCHESTER

AVERY, ELROY J. .....11 N. Goodman St., ALR
BARBER, F. .....75 S. Fitzhugh St., Op.
BARLOW, ROY A. .....Medical Arts Bldg., 277 Alexander St., ALR
BISHOP, VERNON L. .....277 Alexander St., ALR
CLARK, L. H. .....337 Monroe Ave., OALR
GIPNER, J. F. .....Medical Arts Bldg., Op.
JONES, L. W. .....16 N. Goodman St., OALR
KENNEDY, ELDRED WESTON .....Gas and Electric Bldg., Op.
LERNER, MACY LEVI .....332 Park Ave., Op.
MCDOWELL, N. D. .....275 Alexander St., OALR
MORRIS, A. G. .....Medical Arts Bldg., ALR
NASH, C. STEWART .....277 Alexander St., ALR
SHAPERO, I. M. .....289 Alexander St., OALR
SNELL, ALBERT C. .....53 S. Fitzhugh St., Op.

RYE

SOMBERG, J. S. .....127 Overdale Road, Greenhaven, Op.

SARANAC LAKE

BECK, ROBERT J. .....105 Main St., Op.

SYRACUSE

BRITTEN, G. S. .....Medical Arts Bldg., ALR
BROWN, MORTIMER G. .....713 E. Genesee, ALR
BRUST, H. O. .....820 S. Crouse Ave., ALR
CAVE, LINUS S. .....221 Physicians Bldg., ALR
FOWLER, S. R. .....University Block, ALR
GILLETTE, D. F. .....State Tower Bldg., Op.
HALL, EDWIN P. .....State Tower Bldg., ALR
HOOPLE, GORDON D. .....Medical Arts Bldg., ALR
JOY, HAROLD HENRY .....504 State Tower Bldg., Op.
KAUFFMAN, A. B. .....Chimes Bldg., ALR
KLINE, H. G. .....State Tower Bldg., ALR
LEVY, HARRY H. .....State Tower Bldg., Op.
MARLOW, SEARLE B. .....1004 State Tower Bldg., Op.

MOORE, ROY S. .....Medical Arts Bldg., ALR
O.CONNOR, FREDERICK J. .....Medical Arts Bldg., ALR
RUBENSTEIN, A. H. .....Medical Arts Bldg., ALR
WEBSTER, F. R. .....720 State Tower Bldg., Op.

TARRYTOWN

NEWLANDS, W. ALEXANDER .....19 S. Broadway, ALR

TROY

SULZMAN, FRANK MALCOLM .....1831 Fifth Ave., OALR

UTICA

BEATTIE, WILLIAM H. .....258 Genesee St., OALR
FARRELL, JAMES I. .....250 Genesee St., OALR
VALONE, FRANK H. .....258 Genesee St., Op.

WATERTOWN

ATKINSON, WALTER S. .....168 Sterling St., Op.
HENDERSON, L. E. .....168 Sterling St., Op.
MCASKILL, JAS. E. .....Woolworth Bldg., ALR

WHITE PLAINS

MORIARTY, RICHARD W. .....Medical Center, OALR
WOOD, ED. C. .....80 Maple Ave., Op.

WOODHAVEN

FROTHINGHAM, EDWARD .....8644 94th St., OALR

YONKERS

WOLCOTT, CHAS. C. .....27 Ludlow St., ALR

NORTH CAROLINA

ASHEVILLE

GREENE, JOSEPH BERRY .....Haywood Bldg., ALR
SWANN, CECIL C. .....706 Flatiron Bldg., ALR

CHARLOTTE

MATHESON, J. P. .....106 W. 7th St., ALR
PEELER, C. N. .....106 W. 7th St., OALR
SLOAN, HENRY L. .....106 W. 7th St., Op.
WHISNANT, A. M. .....517 Professional Bldg., OALR

DURHAM

ANDERSON, WM. B. .....McPherson Hospital, Op.
MCPHERSON, SAMUEL D. .....McPherson Hospital, ALR

GREENSBORO

REAVES, W. P. .....Reaves Infirmary, 117 W. Sycamore St., OALR

KINSTON

PEERY, VANCE PRICE .....Medical Arts Bldg., ALR

MOREHEAD CITY

JOHNSON, OTIS H. ..... OALR

NEW BERN

DANIELS, R. L. ..... Box 685, OALR

RALEIGH

HICKS, VONNIE MONROE ..... Professional Bldg., Op.

WINSTON-SALEM

DAVIS, THOMAS W. ..... 210 O'Hanlon Bldg., OALR

NORTH DAKOTA

BISMARCK

BUCKINGHAM, TRACY W. ..... 405 Broadway, ALR
CONSTANS, GEORGE MAURICE ..... Clinic Bdlg., Op.

FARGO

LARSON, G. ARTHUR ..... A.O.U.W. Bldg., Op.
RINDLAUB, ELIZABETH P. ..... de Lendrecie Bldg., OALR
RINDLAUB, JOHN H. ..... de Lendrecie Bldg., OALR

MINOT

LAMPERT, M. T. ..... Northwest Clinic, ALR
MCCANNEL, A. D. ..... 15A Central Ave. W., OALR

OHIO

AKRON

ARBUCKLE, DAVID S. ..... 513 W. Market St., OALR
BROWN, L. E. ..... Second Nat'l Bank Bldg., OALR
BRUMBAUGH, JOHN D. ..... Second Nat'l Bank Bldg., OALR
MOORE, T. K. ..... Second Nat'l Bank Bldg., OALR
PARRY, THAYER L. ..... Second Nat'l Bank Bldg., ALR
SEIDEL, U. D. ..... Second Nat'l Bank Bldg., ALR

ALLIANCE

KING, GEORGE L., JR. ..... 537 E. Market St., Op.
KING, G. L., SR. ..... 537 E. Market St., OALR

BELLEFONTAINE

BUTLER, ROBERT H. ..... 112 W. Columbus Ave., OALR
HARBERT, JOHN P. ..... 135 N. Main St., OALR

CANTON

HOFFMANN, JOHN N. ..... 309 Brant Bldg., ALR
TOOT, JOSEPH F. ..... First Nat'l Bank Bldg., Op.
WEAVER, HOMER V. ..... 228 Cleveland Ave., N.W., ALR

CINCINNATI

ABRAHAMSON, IRA R. .....925 Fifth Third Union Trust Bldg., Op.
ASBURY, MARY K. .....2466 Grandin Road, Op.
BEYER, A. G. .....Doctors Bldg., OALR
BROWN, ALBERT LOUIS .....Doctors Bldg., Op.
BRYANT, BEN L. .....19 Garfield Place, ALR
FISCHBACH, VICTOR W. .....Union Central Bldg., ALR
GOODYEAR, H. M. .....Doctors Bldg., ALR
HANS, C. L. .....Provident Bank Bldg., OALR
HARRIS, W. C. .....Groton Bldg., ALR
HENDRICKS, LOUIS J. .....210 W. 12th St., Op.
HOFLING, CHARLES A. .....707 Race St., Op.
HOWARD, ROBERT E. .....Union Central Bldg., ALR
IGLAUER, SAMUEL .....707 Race St., ALR
JONES, C. C. .....Doctors Bldg., ALR
KING, CLARENCE .....Union Central Bldg., Op.
KING, ED. .....1002 Carew Tower, ALR
LOWENTHAL, GERSON .....19 Garfield Place, ALR
LYLE, DONALD J. .....909 Carew Tower, Op.
MITHOEFER, W. .....Doctors Bldg., ALR
PHINNEY, F. D. .....22 W. 7th St., OALR
RAY, VICTOR, JR. .....Carew Tower, Op.
REID, H. W. .....Doctors Bldg., Op.
SELTZ, SAMUEL .....Provident Bank Bldg., ALR
SIEGEL, FRANCIS X. .....610 Union Central Life Bldg., OALR
STANBERY, HENRY .....566 Doctors Bldg., Op.
STEWART, T. M. .....901 Union Trust Bldg., OALR
STITT, H. L. .....656 Doctors Bldg., ALR
STOLL, KARL LUDWIG .....19 West 7th St., OALR
SWING, FRANK U. .....Doctors Bldg., ALR
TANGEMAN, H. F. .....606 Union Central Life Bldg., ALR
THOMPSON, E. H. .....19 W. 7th St., OALR
URNER, M. H. .....2600 Union Central Bldg., OALR
VAIL, D. T. .....Carew Tower, 441 Vine St., Op.
VAIL, H. H. .....Carew Tower, 441 Vine St., ALR

CLEVELAND

ABBOTT, W. J. .....Carnegie Medical Bldg., OALR
BERNS, CHARLES .....Osborn Bldg., ALR
BRUNER, ABRAM BRENEMAN .....1214 Guardian Bldg., OALR
BRUNER, WM. E. .....Guardian Bldg., Op.
BUCHER, WALTER M. .....4187 Pearl Road, ALR
BURKE, T. A. .....Rose Bldg., OALR
CHAMBERLAIN, WEBB PARKS .....7405 Detroit Ave., Op.
CUTLER, FRANKLIN E. .....Hanna Bldg., ALR
DIRION, JOSEPHINE KOOB .....Rose Bldg., Op.
DIXON, FRED WILLIS .....1029 Rose Bldg., ALR
ENGLER, CLARENCE WM. .....2323 Prospect Ave., ALR
FORSYTHE, S. T. .....Rose Bldg., OALR

GARRETT, ERNEST W. ..... Hanna Bldg., ALR
JACOBY, MARK W. ..... Hanna Bldg., Op.
KENDALL, M. RAYMOND ..... 2474 S. Taylor Road, ALR
KINNEY, CHARLES ED. ..... 10515 Carnegie Ave., ALR
LARGE, S. H. ..... Hanna Bldg., ALR
LEMEL, SAMUEL L. ..... 1148 Euclid Ave., ALR
McDONALD, C. L. ..... 964 Rose Bldg., OALR
METZ, ROY B. ..... Hanna Bldg., Op.
METZENBAUM, MYRON ..... Carnegie Medical Bldg., ALR
MITCHELL, HORACE E. ..... 14805 Detroit Ave., ALR
MONSON, S. H. ..... 1322 B. F. Keith Bldg., OALR
MOORE, PAUL G. ..... Republic Bldg., Op.
MOTTO, M. PAUL ..... Rose Bldg., Op.
MUSSUN, W. G. ..... Medical Arts Bldg., ALR
NELSON, CHAS. F. ..... 606 Schofield Bldg., OALR
PITKIN, C. E. ..... Carnegie Medical Bldg., ALR
PRENDERGAST, D. A. ..... Medical Arts Bldg., OALR
PUTNAM, F. I. ..... 1401 Prospect Ave., OALR
QUITTNER, S. S. ..... 10515 Carnegie Ave., OALR
RIEGER, W. H. ..... Hanna Bldg., ALR
ROSENBERGER, HARRY CLIFFORD ..... 10515 Carnegie Ave., ALR
RUEDEMANN, ALBERT DARWIN ..... Cleveland Clinic, Op.
RUST, E. G. ..... Wade Park Manor, E. 107th and Park Lane, Op.
SHIRAS, HOWARD H. ..... 624 Guardian Bldg., Op.
SHIREY, O. M. ..... Osborn Bldg., ALR
SIMONDS, O. F. ..... Hanna Bldg., Op.
STOTTER, ARTHUR L. ..... 1148 Euclid Ave., OALR
TUCKERMANN, W. C. ..... 733 Osborn Bldg., Op.
TUCKERMANN, W. H. ..... 733 Osborn Bldg., OALR
WAUGH, J. M. ..... Cleveland Clinic, Euclid Ave. at 93rd St., OALR
WEIDLEIN, IVAN F. ..... 10515 Carnegie Ave., ALR

COLUMBUS

BEATTY, H. G. ..... 681 E. Broad St., ALR
BROWN, JOHN E. ..... 370 E. Town St., OALR
CLARK, I. G. ..... 188 E. State St., OALR
DAVIES, MORGAN C. ..... 269 E. State St., Op.
DAVIS, WM. C. ..... 327 E. State St., Op.
FROST, ALBERT DALBEY ..... 681 E. Broad St., Op.
HARRIS, ED. WM. ..... 681 E. Broad St., ALR
HAUER, A. M. ..... 327 E. State St., ALR
HELFRICH, ED. D. ..... 269 E. State St., ALR
JOHNSON, DAVID M. ..... 327 E. State St., ALR
MEANS, R. G. ..... 40 S. Third St., ALR
PERRY, CLAUDE S. ..... 40 S. Third St., Op.
POSTLE, C. D. ..... 240 E. State St., ALR
PROUT, A. W. ..... 380 E. Town St., OALR
SANOR, DANIEL G., JR. ..... 206 E. State St., ALR
SILBERNAGEL, C. E. ..... 247 E. State St., OALR

STOUTENBOROUGH, WM. A. ..... 21 E. State St., OALR
SULZER, G. A. ..... 327 E. State St., OALR
THOMAS, FRANCIS W. ..... 327 E. State St., OALR
TIMBERMAN, ANDREW ..... Beggs Bldg., OALR

DAYTON

BINKLEY, ROY S. ..... Fidelity Bldg., OALR
BLACKBURN, W. J. ..... 663 Reibold Bldg., OALR
CULLER, ARTHUR M. ..... Fidelity Medical Bldg., Op.
DRISCOLL, FREDERICK JAS. ..... Reibold Bldg., ALR
DUTROW, H. V. ..... 1040 Fidelity Medical Bldg., OALR
FARMER, A. G. ..... Fidelity Medical Bldg., OALR
FOUTS, JOHN D. ..... Fidelity Medical Bldg., OALR
HARRIS, H. B. ..... Fidelity Medical Bldg., OALR
KILBOURNE, P. H. ..... Fidelity Medical Bldg., OALR
MILLETTE, J. W. ..... 117 S. Main St., OALR

FREMONT

RIDDELL, MALCOLM M. ..... 112½ Front St., ALR

LAKEWOOD

MERICA, F. W. ..... 14805 Detroit Ave., ALR
MILLER, G. LESLIE ..... 14805 Detroit Ave., Op.

LIMA

STUEBER, F. G. ..... 209 W. North St., OALR
STUEBER, PAUL J. ..... 209 W. North St., OALR

LORAIN

MONOSMITH, O. B. ..... O. P. S. Bldg., OALR

MANSFIELD

GARBER, J. M. ..... 14 N. Mulberry St., OALR
WISE, RALPH C. ..... News Bldg., OALR

MARIETTA

SAUER, W. W. ..... 231 Third St., OALR

MARION

BRICKLEY, DANIEL W. ..... Uhler-Phillips Bldg., OALR

MASSILLON

SCOTT, MERLE E. ..... First Nat'l Bank Bldg., OALR

MIDDLETOWN

WILLIAMS, WALTON H. ..... Castell Bldg., OALR

NEWARK

HATCH, C. B. ..... 3 W. Church St., OALR

SALEM

KING, LOWELL W. ..... 470 E. State St., ALR

SANDUSKY

KRAMER, JULIUS C. ..... 130 E. Market St., ALR

SPRINGFIELD

EASTON, J. C. ..... First Nat'l Bank Bldg., OALR
HARTLEY, F. A. ..... 302 E. High St., OALR
HOGUE, D. W. ..... 34 W. High St., OALR
MINOR, C. L. ..... First Nat'l Bank Bldg., OALR

STEUBENVILLE

GOURLEY, G. F. ..... Sinclair Bldg., OALR

TIFFIN

PORTER, E. H. ..... 85 Madison St., OALR

TOLEDO

ALDERDYCE, WM. W. ..... 513 Madison Ave., OALR
BOICE, RALPH E. ..... 230 Michigan St., Op.
KING, CHAS. R. ..... Spitzer Bldg., OALR
LUKENS, CHARLES ..... 316 Michigan St., OALR
LUKENS, JOHN A. ..... 316 Michigan St., ALR
NIPPE, LEONARD ..... 316 Michigan St., OALR
SNYDER, W. H. ..... 2001 Colingwood Ave., OALR
STEINFELD, A. L. ..... 125 Fifteenth St., OALR
WINGER, IRA B. ..... 225 Michigan St., OALR

VAN WERT

EDWARDS, STEPHEN A. ..... Home Guard Bldg., ALR

WARREN

STIMSON, GEORGE W. ..... Mahoning Bldg., OALR

XENIA

MADDEN, REED ..... Allen Bldg., OALR
SHIELDS, LAWRENCE ..... Allen Bldg., ALR

YOUNGSTOWN

EVANS, WILLIAM H. ..... Dollar Bank Bldg., ALR
GOLDCAMP, ED. C. ..... Dollar Bank Bldg., ALR
HARTZELL, SOL. M. ..... Dollar Bank Bldg., OALR
KEYS, JOHN E. L. ..... 613 Home Savings and Loan Bldg., Op.
WALKER, ORVILLE J. ..... Home Savings and Loan Bldg., ALR

ZANESVILLE

CULBERTSON, L. R. ..... Masonic Temple, OALR

OKLAHOMA

ENID

PIPER, ARTHUR S. .....610 S. Monroe, OALR

GUTHRIE

BARKER, CHAS. B. .....224½ W. Okla. Ave., OALR

McALESTER

DAVIS, J. E. .....24½ E. Choctaw, OALR

MUSKOGEE

FULLENWIDER, C. M. .....Barnes Bldg., OALR

OKLAHOMA CITY

FERGUSON, E. S. .....902 Medical Arts Bldg., OALR
GUTHRIE, A. L. .....1200 N. Walker St., OALR
MACDONALD, J. C. .....301 N. W. 12th St., ALR
McHENRY, D. D. .....Medical Arts Bldg., OALR
McHENRY, L. CHESTER .....Medical Arts Bldg., ALR

TULSA

BRASWELL, JAMES C. .....Medical Arts Bldg., OALR
COOK, W. ALBERT .....Medical Arts Bldg., OALR
HARALSON, CHARLES HERNDON .....Medical Arts Bldg., Op.
HUBER, WALTER A. .....Medical Arts Bldg., Op.
ROTH, ALBERT W. .....Medical Arts Bldg., OALR
SMITH, RURIC N. .....Medical Arts Bldg., ALR

OREGON

BAKER

GORDON, GLENN G. .....Alexander Bldg., ALR

EUGENE

NORTON, ARTHUR H. .....1162 Williamette, Op.

LA GRANDE

BOUVY, L. B. .....Bouvy Eye and Ear Hospital, OALR
RALSTON, FRANK L. .....Sacajawea Annex, ALR

PORTLAND

AINSLIE, GEORGE .....Selling Bldg., OALR
BOUVY, HARRY M. .....921 S. W. Washington, St., OALR
BROWNING, ANDREW J. .....Medical Arts Bldg., Op.
CARRUTH, HOWARD ERNEST .....Medical Arts Bldg., OALR
DAVIS, RALPH F. .....Medical Arts Bldg., OALR
FENTON, RALPH A. .....806 Medical Arts Bldg., OALR
HENTON, G. E. .....720 S. W. Washington St., OALR
JOHNSTON, WILSON .....1927 N. W. Lovejoy St., OALR

KARKEET, ROBERT B. .....Medical-Dental Bldg., OALR
KIEHLE, F. A. .....1020 S. W. Taylor St., OALR
KISTNER, F. B. .....600 Mayer Bldg., OALR
LUCAS, HAROLD R. .....Medical-Dental Bldg., OALR
LUPTON, IRVING MARTIN .....806 Medical Arts Bldg., OALR
PETHERAM, C. C. .....Medical Arts Bldg., ALR
TAYLOR, EDGAR M. .....Medical Arts Bldg., Op.
WEEKS, JOHN E. .....360 Edgecliff Road (Retired), Op.

SALEM

BAUM, WM. W. .....Guardian Bldg., ALR
CLEMENT, L. O. .....Guardian Bldg., OALR

PENNSYLVANIA

ALLENTOWN

UHLER, S. MANN .....104 N. Eighth St., ALR

ALTOONA

BUZZARD, JOSIAH F. .....1110 Thirteenth Ave., Op.
GLOVER, S. P. .....1118 Twelfth Ave., OALR

ARDMORE

BOSTWICK, D. S. .....106 Linwood Ave., ALR
FURLONG, THOS. F., JR. .....Times Medical Bldg., ALR

BEAVER

CRUMRINE, NORMAN ROSS .....595 Third St., OALR
SCROGGS, J. J. .....218 E. Third St., Op. A

BETHLEHEM

CHASE, W. D. .....230 E. Broad St., ALR

BUTLER

DOANE, L. L. .....357 N. Main St., OALR
HAZLETT, L. R. .....Savings and Trust Bldg., OALR

CHESTER

CROSS, G. H. .....525 Welsh St., Op.
ORR, ADRIAN V. B. .....525 Welsh St., ALR

CONNELLSVILLE

HARRISON, FRED H. .....308 First Nat'l Bank Bldg., OALR
MCLELLAN, THOMAS G. .....First Nat'l Bank Bldg., Op.

DANVILLE

NEBINGER, REID .....Geisinger Memorial Hospital, ALR

DU BOIS

BEALS, C. WEARNE .....41 N. Brady St., ALR

EASTON

BLOOM, C. HUGH .....65 N. Third St., ALR

EMLENTON

FERMAN, JOHN W. .....OALR

ERIE

DELANEY, JAS. H. .....138 W. 9th St., Op.
DENNIS, EDWARD .....309 W. 10th St., OALR
FUST, JOHN H. E. .....231 W. 7th St., OALR
JACKSON, J. D. .....232 W. 8th St., OALR
RUSSELL, J. A. MERLE .....813 Sassafras St., OALR
SCHLINDWEIN, G. WM. .....138 W. 9th St., OALR
SHREVE, OWEN M. .....162 W. 8th St., Op.

FRANKLIN

JOBSON, G. B. .....West Park and Liberty St., OALR

GREENSBURG

FAIRING, JOHN W. .....118 N. Main St., OALR
MCKEE, CLAUD W. .....Coulter Bldg., OALR

HARRISBURG

DAILEY, GILBERT L. .....618 N. 3rd St., OALR
FARNSLER, H. HERSHEY .....1438 Market St., OALR
GRACEY, GEORGE F. .....209 State St., OALR

HAZLETON

REICHE, O. C. .....Markle Bank Bldg., Op.

HUNTINGDON

HUNT, WILLIAM T., JR. .....514 Penn St., ALR

JOHNSTOWN

BARKER, O. G. A. .....804 Johnstown Bank and Trust Bldg., Op.
GRIFFITH, HAROLD M. ..804 Johnstown Bank and Trust Bldg., Op.
HARRIS, C. M. .....604 Johnstown Bank and Trust Bldg., OALR

LANCASTER

LEFEVRE, WILLIS H. .....234 N. Duke St., ALR

LANSDOWNE

BUSLER, HOWARD S. ....N.E. cor. Baltimore and Owen Aves., ALR

MIDDLETOWN

GEORGE, HENRY W. .....19 N. Union St., Op.

NEW CASTLE

JAQUISH, CHAS. J. .....211 E. Wallace Ave,. ALR

NEW KENSINGTON

STEIM, J. M. .....First Nat'l Bank Bldg., Op.

NORRISTOWN

BEIDEMAN, JOSEPH ELLSWORTH....Montgomery Trust Arcade, Op.
PRICE, JOHN BEADLE .....824 De Kalb St., ALR

PHILADELPHIA

ADLER, FRANCIS HEED .....313 S. 17th St., Op.
ALEXANDER, GEORGE J. .....1930 Chestnut St., ALR
ANNON, W. T. .....235 S. 15th St., ALR
APPLEMAN, L. F. .....308 S. 16th St., Op.
BABBITT, JAMES A. .....1912 Spruce St., ALR
BAER, BENJAMIN F., JR. .....1927 Spruce St., Op.
BALENTINE, P. L. .....1524 Chestnut St., Op.
BATSON, OSCAR V. ....Graduate School of Med., Univ. of Pa., ALR
BLAIR, MORTIMER W. .....N.E. Cor. 20th and Chestnut St., Op.
BOEHRINGER, H. WINFIELD .....700 Central Medical Bldg., OALR
BRICKER, SACKS .....3701 N. Broad St., ALR
BROWN, SAMUEL HORTON .....1930 Chestnut St., Op.
BRUMM, SETH A. .....Medical Arts Bldg., OALR
BURNS, L. J. .....1930 Chestnut St., ALR
BUTT, MIRIAM M. .....2045 Chestnut St., Op.
CARPENTER, C. .....1930 Chestnut St., Op.
CLERF, LOUIS HENRY .....1530 Locust St., ALR
COATES, GEORGE M. .....1721 Pine St., ALR
COHEN, HERMAN B. .....The Lenox, Spruce and 13th Sts., ALR
COHEN, SAMUEL .....2009 Pine St., ALR
COWAN, ALFRED .....1930 Chestnut St., Op.
CREIGHTON, WM. J. .....Physicions'Bldg., Op.
DAVIS, J. LESLIE .....135 S. 18th St., ALR
DAVIS, T. CARROLL .....3128 N. Broad St., ALR
DAVIS, WARREN B. .....135 S. 18th St., ALR
DEICHLER, LYN W. .....1930 Chestnut St., OALR
DINTENFASS, HENRY .....1305 Spruce St., ALR
EMBERY, FRANK .....4801 Penn St. (Frankford) ALR
ERSNER, MATTHEW S. .....1915 Spruce St., ALR
EVES, C. C. .....1910 Spruce St., ALR
FEWELL, A. G. .....1924 Pine St., Op.
FRY, W. E. .....1912 Spruce St., Op.
GIBSON, GLEN G. .....3701 N. Broad St., Op.
GODDARD, H. M. .....1531 Spruce St., ALR
GORDON, WM. .....1721 Pine St., ALR
GOUTERMAN, JOSEPH I. .....1930 Chestnut St., Op.
GOWEN, FRANCIS V. .....904 E. Chelton Ave., ALR
GRIMES, CHAS. H. .....3546 N. Broad St., ALR
GRISCOM, J. MILTON .....255 S. 17th St., Op.
HAWMAN, ERLE G. .....4051 N. Broad St., Op.
HEED, C. R. .....1205 Spruce St., Op.

HEWSON, WM. .....6013 Greene St., Germantown, ALR
HIRST, OTTO C. .....255 S. 17th St., ALR
HOLLOWAY, THOMAS B. .....1819 Chestnut St., Op.
HOUSER, KARL M. .....2010 Spruce St., ALR
HUNTER, R. J. .....2011 Chestnut St., ALR
HUSIK, D. N. .....1930 Chestnut St., ALR
IVY, ROBERT H. .....1930 Chestnut St., ALR
JACKSON, CHEVALIER .....3432 N. Broad St., L
JACKSON, CHEVALIER L. .....3432 N. Broad St., L
KAUFMAN, A. SPENCER .....1923 Spruce St., ALR
KELLY, GEORGE F. J. ..Suite 700, N.E. Cor. 20th and Chestnut, Op.
KRAUSS, FREDERICK .....1930 Chestnut St., OALR
LAESSLE, HENRY A. .....5900 Market St., ALR
LAWRENCE, GRANVILLE A. .....Medical Arts Bldg., Op.
LEWIS, FIELDING O. .....259 S. 17th St., ALR
LILLIE, WALTER I. .....3701 N. Broad St., Op.
LOFTUS, JOHN EDWARD .....1930 Chestnut St., ALR
LUKENS, ROBERT McD. .....1923 Spruce St., ALR
LUONGO, R. A. .....1713 Pine St., ALR
MCANDREWS, LEO F. .....1930 Chestnut St., Op.
MACKENZIE, GEORGE W. .....269 S. 19th St., OALR
MANN, BENJAMIN H. ....N.E. Corner 20th and Chestnut Sts., Op.
MENDEL, JAS. H. .....Germantown Prof. Bldg., ALR
MILLER, M. VALENTINE. ...114 W. Phil-Ellena St., Mt. Airy, ALR
MITCHELL, E. K. .....710 W. Lehigh Ave., ALR
MOORE, WILLIAM FREDERICK .....255 S. 16th St., ALR
O'BRIEN, THOMAS A. .....1727 W. Girard Ave., Op.
OLSHO, S. L. .....235 S. 15th St., OALR
PERSKY, ABRAM H. .....1922 Spruce St., ALR
PETER, L. C. .....1930 Chestnut St., Op.
PONTIUS, P. J. .....1831 Chestnut St., Op.
REESE, WARREN S. .....230 S. 21st St., Op.
RIDPATH, ROBERT F. .....N.E. Cor. 18th and Chestnut Sts., LR
ROMMEL, JOHN C. .....3701 N. Broad St., Op.
RUDOLPHY, JAY BESSON .....1924 Pine St., Op.
SARTAIN, PAUL J. .....2006 Walnut St., Op.
SCARLETT, HUNTER W. .....230 S. 21st St., Op.
SCHWEINITZ, GEORGE E. DE .....1705 Walnut St., Op.
SENER, WALTER J. .....Physicians Bldg., Op.
SHANNON, C. E. G. .....1930 Chestnut St., Op.
SHUSTER, BENJAMIN HARRISON .....1930 Chestnut St., ALR
SKILLERN, S. R., JR. .....1734 Pine St., ALR
SLOANE, HENRY O. .....1717 Pine St., Op.
SLOTKIN, HERMAN B. .....311 S. 20th St., ALR
SPAETH, EDMUND B. .....1930 Chestnut St., Op.
SPENCER, W. H. .....1830 S. Rittenhouse Sq., ALR
STILES, CHAS. M. .....1831 Chestnut St., Op.
STOUT, PHILIP S. .....269 S. 19th St., ALR
TASSMAN, ISAAC S. .....Medical Arts Bldg., Op.

TOWSON, CHAS. E.
Germantown Prof. Bldg., Greene and Coulter Sts., ALR
TUCKER, GABRIEL .....326 S. 19th St., B
WAGERS, ARTHUR J. .....1429 Spruce St., ALR
WEINSTEIN, M. A. .....1309 Spruce St., ALR
WHELAN, G. L. .....2100 Walnut St., ALR
WHELAN, WILLIAM F. .....2015 Walnut St., ALR
WILLIAMS, HORACE J. .....5908 Greene St., Germantown, ALR
ZENTMAYER, WILLIAM .....1930 Chestnut St., Op.

PITTSBURGH

BERNATZ, C. F. .....Park Bldg., Op.
BILLINGS, ROBERT J. .....500 Penn Ave., Op.
BLAIR, W. W. .....121 University Pl., Op.
CARSON, W. E. .....Jenkins Arcade Bldg., Op.
CONWAY, JOHN M. .....5151 Jenkins Arcade, ALR
COOPER, ROY C. .....510 S. Aiken Ave., ALR
CURRY, GLENDON E. .....Westinghouse Bldg., Op.
DAY, KENNETH M. .....121 University Pl., ALR
DAY, EWING W. .....121 University Pl., OALR
FISCHER, N. ARTHUR .....623 Union Trust Bldg., ALR
GROSS, JULIUS E. .....3401 Fifth Ave., ALR
GUERINOT, A. J. .....604 Westinghouse Bldg., OALR
HAGEMANN, JOHN A. .....Highland Bldg., ALR
HAMPSEY, A. R. .....806 May Bldg., ALR
HECKEL, EDWARD B. .....500 Penn. Ave., Op.
JENNINGS, CHAS. W. .....Hiland Bldg., Op.
KREBS, ADOLPH .....Jenkins Arcade Bldg., Op.
LINN, J. G. .....Jenkins Arcade Bldg., Op.
MCCOLLOUGH, THOMAS B. .....121 University Pl., ALR
MCCREADY, J. HOMER .....121 University Pl., ALR
MACLACHLIN, A. A. .....May Bldg., ALR
MARKEL, J. CLYDE .....Westinghouse Bldg., Op.
MARSHALL, WATSON .....121 University Pl., ALR
MEANOR, WM. C. .....1715 Clark Bldg., Op.
MOYER, JOHN S. .....303 Coltart Ave., ALR
PATTERSON, ELLEN J. .....121 University Pl., ALR
PLUMER, JOHN S. .....121 University Pl., Op.
REED, CHAS. L. .....Jenkins Bldg., Op.
RIMER, FRANK H. .....Jenkins Arcade, ALR
SEEGMAN, SIMON .....Medical Bldg., ALR
SHUMAN, G. H. .....Park Bldg., Op.
SIMPSON, JOHN REID .....Medical Arts Bldg., ALR
STIEREN, EDWARD .....626 Union Trust Bldg., Op.
TAYLOR, M. C. .....May Bldg., Op.
THORPE, H. E. .....8075 Jenkins Arcade Bldg., Op.
TODD, GROVER CLEVELAND .....Jenkins Arcade Bldg., ALR
TURNER, H. H. .....Clark Bldg., Op.
WEISSER, E. A. .....806 May Bldg., Op.

WIBLE, LEROY E. ..... Empire Bldg., ALR
YOST, WALTER MILTON ..... Westinghouse Bldg., Op.

POTTSVILLE

CRESS, WILLIAM J. ..... 318 W. Market St., ALR

READING

CRAIG, PAUL C. ..... 220 N. 6th St., Op.
HIGH, ISAAC B. ..... 326 N. 5th St., ALR
LANDIS, JAMES E. ..... 517 Walnut St., ALR
RHODE, SOLON L. ..... 238 N. 6th St., OALR

RIDGWAY

MCALLISTER, J. C. ..... 10 S. Mill Ave., AL

ROCHESTER

YOST, WALTER M. ..... Peoples Bank Bldg., Op.

SAYRE

WEINBERGER, NELSON S. ..... Robert Packer Hospital, OALR

SCRANTON

CORSER, J. B. ..... 345 Wyoming Ave., Op.
CURTIN, EUGENE A. ..... 506 Spruce, OALR
SULLIVAN, J. J., JR. ..... 228 Scranton Life Bldg., ALR
WILLIAMS, M. M. ..... Medical Arts Bldg., Op.

SHENANDOAH

MONAHAN, JAMES J. ..... 31 S. Jordin St., ALR

TURTLE CREEK

HUNTER, JAS. F. ..... 503 Penn Ave., ALR

UPPER DARBY

MOORE, GEO. H. ..... 101 Long Lane and Chestnut St., OALR

WARREN

BALL, MICHAEL V. ..... 316 Hazel St., OALR

WASHINGTON

MCCULLOUGH, CLARENCE JOSEPH .... Washington Trust Bldg., ALR
MCKENNAN, JAMES W. ..... 514 Washington Trust Bldg., Op.
MCMURRAY, J. B. ..... Washington Trust Bldg., OALR

WAYNESBURG

STAHLMAN, FRED C. ..... Commercial Bldg., OALR

WILKES-BARRE

BUCKMAN, E. U. ..... 70 S. Franklin St., OALR
BUCKMAN, LEWIS T. ..... 83 S. Franklin St., ALR

CARR, GEORGE W. .....54 S. Franklin St., OALR
CONNOLE, JOSEPH V. .....Miners Nat'l Bank Bldg., Op.
KISTLER, O. F. .....43 N. Franklin St., Op.AR
MCLAUGHLIN, P. A. .....71 S. Washington St., OALR
MARSDEN, W. C. .....99 N. Franklin St., ALR
SCHAPPERT, N. LOUIS .....59 S. Washington St., OALR

YORK

FACKLER, CHAS. L. .....11 E. Market St., ALR
KLINEDINST, JOHN FRED .....220 S. George St., Op.A

PUERTO RICO

SAN JUAN

FONT, JUAN H. .....Cruz St. No. 13, ALR

RHODE ISLAND

NEWPORT

JACOBY, DOUGLAS P. A. .....106 Touro St., Op.AL

PROVIDENCE

ADAMS, FRANK M. .....122 Waterman St., ALR
BOLOTOW, NATHAN A. .....126 Waterman St., ALR
CAPRON, F. P. .....118 Angell St., OALR
DIMMITT, FRANK W. .....195 Thayer St., OALR
HARVEY, N. D. .....112 Waterman St., OALR
LEECH, J. W. .....167 Angell St., OALR
MESSINGER, H. C. .....210 Angell St., Op.
MUNCY, WM. M. .....162 Angell St., OALR
PORTER, L. B. .....199 Thayer St., OALR
SHARP, BENJ. S. .....339 Thayer St., OALR
TINGLEY, LOUISA PAINE .....171 Westminster St., Op.
WINKLER, HERMAN A. .....224 Thayer St., ALR

SOUTH CAROLINA

CHARLESTON

TOWNSEND, JOHN FERRARS .....85 Wentworth St., OALR

COLUMBIA

ASBILL, DAVID S. P. .....1512 Marion St., Op.
BRISTOW, WALTER J. .....1512 Marion St., ALR

GREENVILLE

CARPENTER, E. W. .....19½ W. North St., OALR
JERVEY, JAMES W. .....101 Church St., OALR

SPARTANBURG

GRAY, E. B. .....909 Montgomery Bldg., ALR

SOUTH DAKOTA

HURON

GROSVENOR, L. N. .....366 Dakota Ave. So., OALR

RAPID CITY

WALSH, JOSEPH MARK .....OALR

SIOUX FALLS

GREGG, JOHN B. .....612 Security Nat'l Bank Bldg., OALR
KELLER, S. A. .....303 S. Minnesota Ave., OALR

WATERTOWN

JOHNSON, A. EINAR .....Kemp and Broadway, OALR

YANKTON

HOHF, JULIUS A. .....Clinic Bldg., OALR

TENNESSEE

CHATTANOOGA

HACKNEY, F. J. .....311 McCallie Ave., OALR
HOGSHEAD, J. M. .....602 Georgia Ave., OALR
LAWWILL, STEWART .....Medical Arts Bldg., OALR
STEELE, WILLARD .....721 Broad St., OALR

CLEVELAND

FERGUSON, E. R. .....P. and S. Hospital, ALR

DYERSBURG

BREWER, JOHN D. .....Braid, Brewer General Hospital, OALR

JOHNSON CITY

JONES, U. G. .....Corner Market and Boone Sts., ALR

KNOXVILLE

KINCAID, J. H. .....421 W. Church Ave., OALR
PATTERSON, R. W. .....Medical Arts Bldg., ALR
POTTER, WILLIAM W. P. .....Medical Bldg., OALR
REAVES, ROBERT G. .....422 W. Cumberland Ave., OALR

MEMPHIS

ANTHONY, DANIEL HARBERT .....1720 Exchange Bldg., Op.
BLASSINGAME, C. D. .....899 Madison Ave., ALR
BLUE, J. B. .....62 N. Main St., OALR
ELLETT, E. C. .....Exchange Bldg., Op.
EVANS, S. S. .....Physicians and Surgeons Bldg., ALR
FARRINGTON, P. M. .....899 Madison Ave., ALR

HARRIS, J. HARLEY ..... Medical Arts Bldg., ALR
HUGHES, J. A. ..... Sterick Bldg., OALR
LEVY, LOUIS ..... Commerce Title Bldg., ALR
LEWIS, A. C. ..... 130 Madison Ave., OALR
LEWIS, PHILIP MERIWETHER ..... Exchange Bldg., OALR
MCKINNEY, R. ..... 899 Madison Ave., ALR
MINOR, H. F. ..... Bank of Commerce Bldg., OALR
QUALLS, HARLEY W. ..... Exchange Bldg., OALR
RYCHENER, RALPH O. ..... 130 Madison Ave., Op.
SANDERS, SAM H. ..... 248 Madison Ave., ALR
SELIGSTEIN, MILTON B. ... 801 Union and Planters Bank Bldg., OALR
SHEA, J. J. ..... 1018 Madison Ave., ALR
SIMPSON, W. LIKELY ..... Phys. and Surg. Bldg., ALR
STANFORD, J. B. ..... 899 Madison Ave., OALR
STINSON, WM. DAVID ..... 805 Medical Arts Bldg., ALR
WATKINS, EDWIN DIAL ..... 1412 Exchange Bldg., OALR

NASHVILLE

BRYAN, JAMES LESLIE ..... 919 Bennie-Dillon Bldg., OALR
CULLOM, M. M. ..... 1119 Bennie-Dillon Bldg., OALR
EZELL, H. C. ..... 706 Church St., OALR
HASTY, F. E. ..... Medical Arts Bldg., ALR
KENNON, W. G. ..... Doctors Bldg., OALR
ORR, EUGENE ..... Doctors Bldg., ALR
SULLIVAN, ROBT. ED. ..... Doctors Bldg., Op.
WARNER, ROBERT JAY ..... 708 Church St., Op.
WILKERSON, WM. W., JR. ..... Bennie-Dillon Bldg., ALR

TEXAS

AMARILLO

CRUME, JOHN JAS. ..... 320 Amarillo Bldg., ALR
DUNCAN, ROBERT AUSTIN ..... 339 Amarillo Bldg., ALR
STREIT, AUGUST J. ..... 811 Fisk Bldg., ALR

AUSTIN

HILGARTNER, H. L., JR. ..... Norwood Bldg., Op.
HILGARTNER, H. L., SR. ..... Norwood Bldg., Op.
KEY, SAM N. ..... Norwood Bldg., OALR
ROBISON, J. T. ..... Norwood Bldg., ALR

BROWNWOOD

ANDERSON, W. B. ..... 309 West Anderson St., OALR

CORPUS CHRISTI

STROUD, EDWARD F. ..... Medical Professional Bldg., ALR

DALLAS

CARY, E. H. ..... Medical Arts Bldg., OALR
DARROUGH, L. E. ..... 4105 Live Oak St., ALR

DECHERD, H. B. .....1515 Medical Arts Bldg., OALR
MARCHMAN, OSCAR M. .....Medical Arts Bldg., ALR
MCLAURIN, J. G. .....Medical Arts Bldg., OALR
MCREYNOLDS, JOHN O. .....907 Mercantile Bank Bldg., OALR
NELSON, LEO A. .....4105 Live Oak St., ALR
NEWTON, F. H. .....907 Mercantile Bank Bldg., OALR
SELLERS, L. M. .....Medical Arts Bldg., ALR
THOMAS, W. MAXWELL .....Medical Arts Bldg., Op.

DEL RIO

SCHULZE, E. C. .....504 First Nat'l Bldg., OALR

EL PASO

IRVIN, EDGAR H. .....903 First Nat'l Bank Bldg., OALR
SCHUSTER, FRANK P. .....First Nat'l Bank Bldg., OALR
SCHUSTER, S. A. .....First Nat'l Bank Bldg., ALR
VANDEVERE, WILLIAM E. .....Bassett Tower, OALR

FORT WORTH

GOODMAN, THOMAS L. .....Medical Arts Bldg., ALR
GOUGH, ROY H. .....Medical Arts Bldg., OALR
HOWARD, EDWARD L. .....405 Medical Arts Bldg., OALR
JACKSON, ATRAS E. .....Medical Arts Bldg., Op.
RICHARDSON, JAMES JOSEPH .....Cook Hosp. and Beall Clinic, ALR
SCHENCK, C. P. .....Medical Arts Bldg., OALR
WARWICK, HAROLD L. .....913 Medical Arts Bldg., ALR

GREENVILLE

WARD, JAMES W. .....2607½ Lee St., ALR

HOUSTON

CODY, CLAUDE C. .....1304 Walker Ave., ALR
DAILY, LOUIS .....1117 Medical Arts Bldg., OALR
DAILY, RAY K. .....1117 Medical Arts Bldg., OALR
DICKSON, J. CHARLES .....Medical Arts Bldg., ALR
ELLES, NORMA B. .....1704 Niels Esperson Bldg., OALR
FOSTER, J. H. .....1304 Walker Ave., ALR
GOAR, EVERETT LOGAN .....1300 Walker Ave., Op.
GRIFFEY, EDWARD W. .....Medical Arts Bldg., ALR
HADEN, HENRY C. .....1914 Travis St., Op.
ISRAEL, SIDNEY .....2010 Niels Esperson Bldg., ALR
LOGUE, LYLE J. .....1304 Walker Ave., ALR
MAXWELL, HARVEY C. .....Station Hosp., Fort Sam Houston, OALR
RALSTON, WALLACE .....1300 Walker Ave., Op.

LONGVIEW

HURST, V. REEVES .....Hurst Hospital, OALR

LUBBOCK

MALONE, F. B. .....1917 Tenth St., ALR

SAN ANGELO

WINDHAM, ROBERT EDWARD.....234 W. Beauregard Blvd., OALR

SAN ANTONIO

ATKINSON, D. T. .....827 Medical Arts Bldg., OALR
BURLESON, J. H. .....1034 Nix Professional Bldg., OALR
GILL, WILLIAM DAVIS .....705 E. Houston St., OALR
PARRISH, ROBERT E. .....Medical Arts Bldg., ALR
ROSEBROUGH, FRANK HENRY .....Moore Bldg., Op.
WESSELS, ANDREW B. .....Nix Professional Bldg., ALR

TEMPLE

MCDAVITT, BERTHA SCHENCK STECKER.....Main and Ave. A, ALR
WOODSON, BURBANK P. .....Woodson Hospital, OALR
WOODSON, WARREN B. .....5th St. and Avenue G, Op.

TEXARKANA

FULLER, T. E. .....209 Pine St., OALR
MANN, ALBERT H. .....209 Pine St., ALR

TURTLE CREEK

HUNTER, JAS. F. .....536 Penn Ave., ALR

TYLER

VAUGHN, E. H. .....Peoples Nat'l Bank Bldg., OALR

WACO

AYNESWORTH, H. T. .....Medical Arts Bldg., OALR
COLGIN, WM. ED. .....605 Columbus Ave., Op.
SPENCER, SHELBY CRUTHIRDS .....Professional Bldg., Op.

WELLINGTON

HIGH, CLIFTON E. .....Small Bldg., Op.

UTAH

HYRUM

GILL, A. E. .....(Retired) OALR

OGDEN

HETZEL, C. C. .....Eccles Bldg., OALR

PROVO

MERRILL, H. G. .....35 East 2nd South St., OALR

SALT LAKE CITY

HAMPTON, ROBERT R. .....804 Boston Bldg., ALR
LE COMPTE, E. D. .....9 Exchange Place, OALR
NEHER, EDWIN MANSON .....Boston Bldg., Op.
SMITH, WM. LEROY .....Medical Arts Bldg., Op.
STAUFFER, F. LEAVER .....Medical Arts Bldg., ALR
STAUFFER, FRED .....Medical Arts Bldg., OALR
WELSH, THOMAS F. .....920 Boston Bldg., OALR
WHITE, VIVIAN P. .....Ezra Thompson Bldg., ALR

VERMONT

BARRE

JARVIS, DE FOREST C. ..... Quarry Bank Bldg., ALR

BURLINGTON

ARNOLD, FRANCIS J. ..... 182 Pearl St., OALR

RUTLAND

MARSHALL, GEORGE GUERIN ..... Gryphon Bldg., OALR

ST. JOHNSBURY

RICKER, WILLIAM G. ..... 33 Main St., Op.

VIRGINIA

DANVILLE

DICKERSON, WM. E. ..... Masonic Temple, ALR

FREDERICKSBURG

HOLLOWAY, JOSEPH M. .. N.W. Cor. Hanover and Charles Sts., ALR

HARRISONBURG

PRESTON, H. GRANT ..... Professional Bldg., ALR

RICHMOND

COURTNEY, ROBERT H. ..... Professional Bldg., Op.
GATEWOOD, E. T. ..... Professional Bldg., ALR
HILL, EMORY ..... 501 E. Franklin St., Op.
HUGHES, T. E. ..... Professional Bldg., ALR
MILLER, CLIFTON, M. ..... Stuart Circle Hospital, OALR

ROANOKE

GILL, E. G. ..... Box 2467, ALR
HAMLIN, FRED EUGENE ..... Medical Arts Bldg., ALR
STONE, HARRY B. ..... Medical Arts Bldg., ALR
YOUNG, CHARLES A. ..... Medical Arts Bldg., Op.

UNIVERSITY

WOODWARD, FLETCHER DRUMMOND ..... Box 1685, ALR

WINCHESTER

MCGUIRE, H. H. ..... 105 N. Braddock St., OALR

WASHINGTON

ABERDEEN

YOUNG, CLARENCE M. ..... 305 S. H St., ALR

BELLINGHAM

HOWE, SPENCER S. ..... Bellingham Nat'l Bank Bldg., ALR

EVERETT

MURPHY, ALBERT B. ..... Medical and Dental Bldg., ALR
SECOY, HARRY R. ..... Everett Clinic, ALR

LONGVIEW

BARTON, JOHN F. ..... 1346 Fourteenth Ave., ALR

SEATTLE

ADAMS, FRED'K W. ..... Roosevelt Clinic, 1114 Boylston Ave., OALR
BELL, WILL OTTO ..... Medical-Dental Bldg., Op.
CHASE, E. FRANK ..... 1008 Cobb Bldg., OALR
DOWLING, J. THOMAS ..... Virginia Mason Hospital, OALR
FAIRCHILD, NORA M. ..... Stimson Bldg., Op.
HOFFMAN, W. F. ..... 1317 Marion St., Op.
MCCOY, L. L. ..... 817 Summit Ave., Op.
SEELYE, W. K. ..... 1215 Fourth Ave., OALR
SHANNON, C. W. ..... New 4th and Pike Bldg., Op.
STILLSON, H. ..... Seabord Bldg., OALR
WURDEMANN, H. V. ..... 320 Stimson Bldg., Op.

SPOKANE

CODD, ALPHONSO N. ..... Paulsen Med. and Dental Bldg., ALR
PARSELL, LOUIS A. ..... Paulsen Med. and Dental Bldg., ALR
ROTT, O. M. ..... Paulsen Med. and Dental Bldg., OALR
SMITH, CARROLL L. ..... Paulsen Med. and Dental Bldg., OALR
VEASEY, C. A., SR. ..... Paulsen Med. and Dental Bldg., OALR
VEASEY, C. A., JR. ..... Paulsen Med. and Dental Bldg., OALR

TACOMA

CAMERON, WM. G. ..... Medical Arts Bldg., OALR
DAVIES, J. S. ..... 2611 N. Union Ave., OALR
HOWE, A. W. ..... Medical Arts Bldg., OALR
PAUL, WM. C. ..... Medical Arts Bldg., ALR

WALLA WALLA

GOWEN, GEORGE ROBERT ..... 306 Baker Bldg., ALR

WEST VIRGINIA

BLUEFIELD

ST. CLAIR, C. T. ..... Corner Bland and Ramsey Sts., ALR

FAIRMONT

HALL, SOBISCA S. ..... 95 Fairmont Ave., ALR
JOHNSON, H. R. ..... 320 Jefferson St., OALR
TRAUGH, GEO. H. ..... 309 Cleveland Ave., ALR

HUNTINGTON

MOORE, J. HALLOCK ..... 1050 Fifth Ave., ALR
MOORE, T. W. ..... 1050 Fifth Ave., OALR

MORGANTOWN

WYLIE, C. B. .....269 High St., OALR

PARKERSBURG

HARTMAN, EUGENE C. .....610½ Market St., OALR

WELCH

SCHIEFELBEIN, HARRY T. .....Stevens Clinic Hospital, Op.

WHEELING

FAWCETT, IVAN .....75 Twelfth St., OALR
JONES, EDMUND LLOYD .....Wheeling Clinic, OALR
TOMASSENE, R. A. .....1144 Market St., OALR
ZUBAK, MATTHEW F. .....Citizens Mutual Trust Bldg., OALR

WISCONSIN

APPLETON

BROOKS, E. H. .....Zuelke Bldg., OALR
RECTOR, A. E. .....103 W. College Ave., OALR

EAU CLAIRE

PAYNE, A. L. .....119 S. Barston St., OALR
TANNER, JAS. WALLACE .....314 E. Grand Ave., ALR

FOND DU LAC

FOLSOM, W. H. .....11 N. Main St., OALR

GREEN BAY

COMEE, W. C. .....Northern Bldg., OALR
NADEAU, E. G. .....Bellin Bldg., OALR

JAYNESVILLE

PEMBER, AUBREY H. .....500 W. Milwaukee St., OALR

KENOSHA

ASHLEY, T. W. .....5711 Eighth Ave., OALR

LA CROSSE

DOUGLAS, FREDERICK ALEXANDER.....303 State Bank Bldg., OALR
SMITH, D. S. .....State Bank Bldg., OALR

MADISON

DAVIS, F. A. .....224 W. Washington Ave., OALR
DIMOND, WALDO B. .....16 N. Carrol St., ALR
NEFF, EUGENE E. .....224 W. Washington Ave., Op.
SCHUBERT, CLARENCE K. .....1 S. Pinckney St., Op.

MANITOWOC

ANDREWS, M. P. .....826 S. 8th St., OALR
SCHNECK, NATHAN .....Commerce Blk., ALR

MARINETTE

MAY, J. V. ..... 1703 Main St., OALR

MARSHFIELD

COPPS, LYMAN ALDEN ..... Marshfield Clinic, OALR

MILWAUKEE

BEEBE, C. S. ..... 324 E. Wisconsin Ave., Op.
BROOK, JEFFREY J. ..... 231 W. Wisconsin Ave., ALR
CHAPMAN, V. A. ..... 324 E. Wisconsin Ave., OALR
GORDON, J. S. ..... 922 Bankers Bldg., OALR
GROVE, W. E. ..... 324 E. Wisconsin Ave., OALR
HAESSLER, F. HERBERT ..... Wells Bldg., Op.
HEEB, HARRY J. ..... 204 W. Wisconsin Ave., OALR
HIGGINS, S. G. ..... 324 E. Wisconsin Ave., OALR
HITZ, H. B. ..... Bankers Bldg., ALR
HITZ, JOHN B. ..... 208 E. Wisconsin Ave., Op.
MCCORMICK, THOMAS F. ..... Wells Bldg., ALR
MARKS, JULIAN BARNETT ..... 735 N. Water St., ALR
MULSOW, J. E. ..... 231 W. Wisconsin Ave., OALR
RYAN, ED. R. ..... 324 E. Wisconsin Ave., Op.
SCHMIDT, HERBERT G. ..... 238 W. Wisconsin Ave., ALR
SPROULE, RALPH P. ..... 208 E. Wisconsin Ave., OALR
TOLAN, THOS. L. ..... 1238 Wells Bldg., ALR
TOMB, E. H. ..... 110 E. Wisconsin Ave., ALR
WALDECK, EDWARD A. ..... 238 W. Wisconsin Ave., Op.

RACINE

HOLMES, BENJAMIN H. ..... 523 Main St., ALR

SHEBOYGAN

NEUMANN, WM. H. ..... Sheboygan Clinic, OALR

SUPERIOR

MYERS, ELMER A. ..... 407 Board of Trade Bldg., OALR
SMITH, R. C. ..... Board of Trade Bldg., OALR

WAUSAU

SCHLEGEL, HERMAN T. ..... 501 Third St., OALR

WHITEHALL

SIMONS, NEAL S. ..... MacCornack Clinic, ALR

WYOMING

CHEYENNE

BECK, FREDERICK L. ..... 408 Hynds Bldg., ALR
STRADER, GEORGE L. ..... 408 Hynds Bldg., OALR

AFRICA

NATAL

DURBAN

JOHNSON, LINDSAY ..... "Fairlawn" Esplanade, Op.

AUSTRIA

VIENNA, VI

BRUNNER, HANS ..... Gumpendorfer St. 25, ALR

CANADA

ALBERTA

EDMONTON

BROWN, W. HAROLD ..... Bank of Montreal Bldg., OALR

BRITISH COLUMBIA

VANCOUVER

BOUCHER, R. B. ..... Medical Dental Bldg., OALR

VICTORIA

KEYS, MORTON J. ..... 409 Pemberton Bldg., OALR

MANITOBA

BRANDON

MCDIARMID, H. OLIVER ..... 35 Clement Blk., OALR

NEW BRUNSWICK

ST. JOHN

CHIPMAN, L. D. V. ..... 42 Coburg St., OALR

NOVA SCOTIA

HALIFAX

CUNNINGHAM, ALLAN R. ..... 260 Barrington St., OALR

ONTARIO

HAMILTON

ARRELL, W. ..... 317 Main St., E., OALR
MACFARLANE, P. B. ..... Medical Arts Bldg., OALR

WELLAND

BRIANT, THOMAS ERNEST ..... 28 East Main St., ALR

OTTAWA

FRASER, W. G. ..... Medical Arts Bldg., OALR

PETERBOROUGH

BUCHANAN, NORMAN D. ..... 129 Murray St., OALR

TORONTO

ALEXANDER, SEEYMAN L. ..... Medical Arts Bldg., ALR
BOCHNER, MAXWELL K. ..... 329 Bloor St., W., Op.
BARNES, LANCELOT ..... 170 St. George St., Op.
BRADLEY, BASIL V. ..... Medical Arts Bldg., ALR
CAMPBELL, A. A. ..... 86 Bloor St. W., ALR
DAVIES, T. A. ..... 578 Sherborne St., OALR
GOLDSMITH, PERRY G. ..... Medical Arts Bldg., ALR
HILL, C. E. ..... 160 Bloor St., W., Op.
MCCULLOCH, ROBERT J. P. ..... Medical Arts Bldg., OALR
MCGREGOR, GREGOR WM. ..... 170 St. George St., ALR
PENTECOST, R. S. ..... 338 Bloor St., E., OALR
RISDON, E. F. ..... Medical Arts Bldg., ALR
SEBERT, L. J. ..... Medical Arts Bldg., Op.
SNELL, GEO. C. ..... Medical Arts Bldg., ALR
STARK, WILLIAM B. ..... Medical Arts Bldg., ALR
SULLIVAN, JOS. A. ..... 170 St. George St., ALR
WRIGHT, WALTER WALKER ..... Medical Arts Bldg., Op.

QUEBEC

MONTREAL

BALLON, D. H. ..... 1471 Crescent St., ALR
BIRKETT, H. S. ..... 1190 Mountain St., ALR
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GRAVES, BASIL .....11 Devonshire Place, Upper Wimpole St., W. 1
KIRKPATRICK, LT.-COL. HENRY.....54 Welbeck St., W. 1
LAWFORD, J. B. .....Sagamore New Road, Ashtead, Surrey
LISTER, SIR WILLIAM T. .....24 Devonshire Pl., W. 1
TILLEY, HERBERT .....University College Hospital

HOLLAND

LEIDEN

VAN DER HOEVE, PROF. J. .....Rijnsburgerweg 6A

HUNGARY

BUDAPEST

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MEXICO

MEXICO CITY

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CANAL ZONE

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CRISTOBAL

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SWITZERLAND

ZURICH

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